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CORRELATION OF POSTMORTEM CHEST TELEROENTGENOGRAMS WITH AUTOPSY FINDINGS

WITH SPECIAL REFERENCE TO PULMONARY EMBOLISM AND INFARCTION*

By AUBREY O. HAMPTON *and* BENJAMIN CASTLEMAN
BOSTON, MASSACHUSETTS

EXACT and detailed confirmation of the interpretations of roentgenograms of the chest is often difficult to obtain for three reasons: First and most important is the fact that, even if an autopsy is performed, one cannot rule out any change that may have occurred between the time of the last roentgenogram and that of death. There is no doubt that during the last few hours of a patient's life a large quantity of fluid may accumulate; bronchopneumonia can become more widespread; hemorrhage into a tumor can increase its size. Quite often correlations between roentgen and pathological findings are unjustly made with roentgenograms taken several days or even weeks before autopsy, despite the extensive changes which can occur during this period. Secondly, if an attempt is made to take roentgenograms shortly prior to death, it is necessary to use a portable machine and the roentgenograms may not show the lesions sharply and accurately. Finally, when the lungs are removed at postmortem they are collapsed, and accurate correlation between the air-distended lung as seen on the roentgenogram and the autopsy specimen is practically impossible. This is especially true as regards distances and locations of lesions. To obviate these difficulties and to leave no loophole for the sometimes undue skepticism on the part of roentgenologists for pathologists who do not find what the former have seen or discover what the roentgenologist has missed, a cooperative method was devised to study the chest.

METHOD

As soon as permission is granted for a postmortem examination a chest roentgenogram is taken of the body at a distance of 7 feet. The body is suspended vertically against a wall, upon which a plate holder is permanently fixed (Fig. 1). The method of suspending the body consists in using a very sturdy leather halter (canvas is not strong enough), the front part of which fits snugly under the chin, with side straps around the face and secured at the back of the neck. The halter is lined with felt so that the face is not marred. The halter and

* From the Departments of Roentgenology and Pathology, Massachusetts General Hospital, Boston. Read at the Thirty-ninth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 20–23, 1938, and before the New England Pathological Society, Feb. 16, 1939.

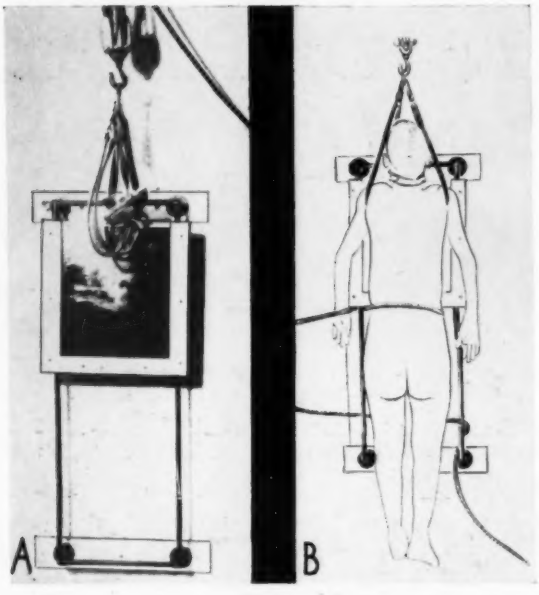

FIG. 1. *A*, the plate holder attached to wall, and leather halter attached to hook and pulley which is firmly bolted to ceiling; *B*, the body is suspended by halter and ropes.

the ropes under the arms which give further support for the body are both attached to a pulley arrangement which is securely attached to the ceiling. Both posteroanterior and lateral roentgenograms are taken. A tube with a fine focus is used and 15 ma-sec. of exposure is given for the posteroanterior view and 45 ma-sec. for the lateral. In cases of suspected pneumothorax or chest wall lesions the autopsy is not begun until the roentgenograms have been interpreted. Usually, however, no further delay in the autopsy is necessary.* At autopsy both lungs are removed together with the trachea, great care being taken not to tear the visceral pleura. Kaiserling solution No. 1 (a formalin fixative) is instilled by gravity through the trachea until both lungs are approximately the same size as they were during life (Fig. 2). The trachea is then clamped and the specimen submerged in a large crock containing the same fixative and allowed to remain for about a week. At the end of this period the lungs are sectioned by the pathologist in the presence of the roentgenologist. An attempt is made to find anatomical explanation for all the shadows seen on the postmortem roentgenograms and conversely the roentgenologist attempts to see on the roentgenogram most of the lesions present in the specimen. We have studied 400 cases according to this method.

THE NORMAL POSTMORTEM ROENTGENOGRAM

After death there is reduction in the size and incomplete aeration of the lung appar-

* Obviously, this method insures having chest roentgenograms on patients who have been too ill, or who have died too soon after entry to have one taken. Occasionally this is important if an unusual or unsuspected chest lesion is discovered at autopsy.

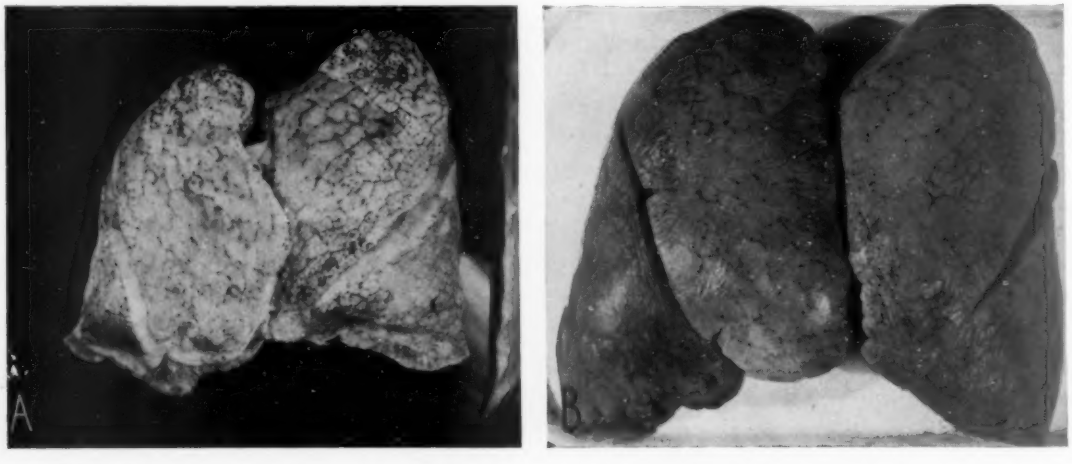

FIG. 2. Lungs before and after injection. Note the smaller size and ill defined character of the deflated and collapsed specimen, *A*, when compared with the inflated, sharply outlined injected lung, *B*.

ently due to narrowing of the intercostal spaces, the high position of the diaphragm, and the contraction of the lung because of its inherent elasticity. The usual postmortem roentgenogram of the chest at close distances with the patient horizontal has often been shown to be unsatisfactory because of incomplete aeration of the lungs and distortion. By suspending the body in the upright position in the manner already described, however, this decreased aeration of the lung is somewhat compensated for by the downward traction of the diaphragm by the abdominal viscera, and by the widening of the intercostal spaces by means of the rope supports under the axillae. The general appearance of the postmortem roentgenogram of the chest corresponds to one taken antemortem during quiet breathing or expiration (Fig. 3). The diaphragm is usually about two interspaces higher than that seen on an inspiration roentgenogram, the upper lobes are fairly well expanded, and the lower lobes are incompletely aerated. The latter fact is particularly noticeable in the lateral view, which also shows a considerable reduction in the posteroanterior diameter of the chest. A comparison of the antemortem and postmortem roentgenograms taken of a patient with normal lungs shows a marked variation in the heart and pulmonary vascular shadows. In the postmortem roentgenogram the blood vessels are usually much smaller and less distinct, the aorta about one-half the diameter, and the heart either dilated or contracted. The chief differences, therefore, in the antemortem and postmortem roentgenograms are the degree of aeration of the lung and the alteration in the size and shape of the cardiovascular shadows.

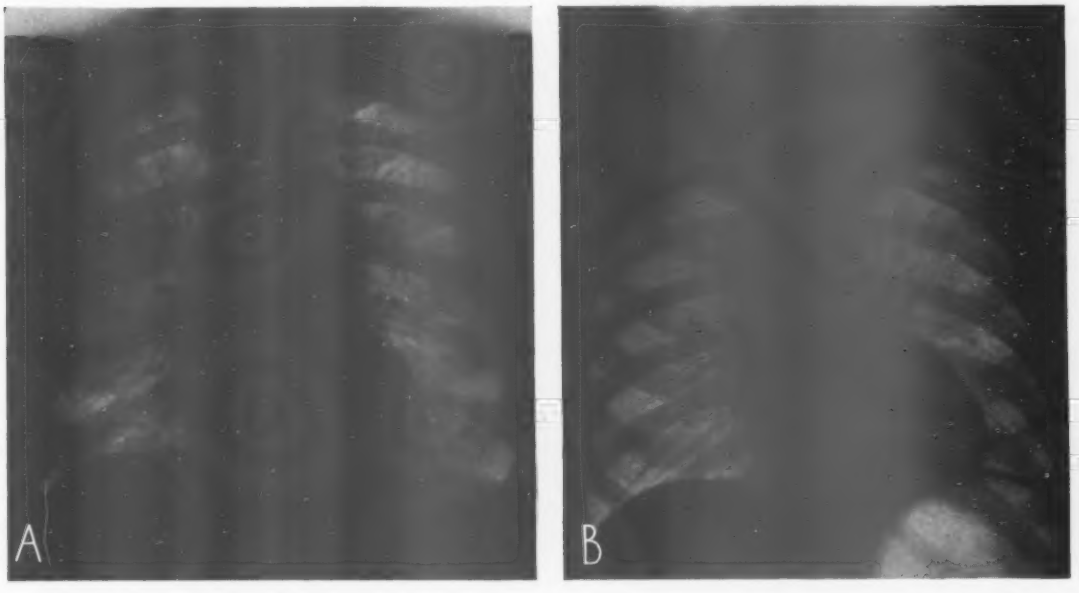

Fig. 3. *A*, antemortem roentgenogram of normal chest. *B*, postmortem roentgenogram, same subject, position and technique. Note elevation of diaphragm and reduction in size of aorta and pulmonary vessels.

PULMONARY EMBOLISM AND INFARCTION

Although there are many potential aspects of a correlative study of this type, in this report we shall deal only with the problem of pulmonary embolism and infarction. As the project advanced it became quickly apparent that many of the elementary features of pulmonary infarction were not understood adequately enough to permit of accurate correlation of roentgenograms,

clinical histories, and anatomic findings, and an extensive anatomic and histologic study proved necessary to provide an adequate background. For this purpose use was made of a larger series of cases drawn from the 3,500 autopsies performed in the laboratory during the ten year period before the combined study was begun. In this larger series either pulmonary embolism or infarction was noted in 9 per cent of the cases, and in 3.5 per cent of this same group it was considered a major cause of death. In the combined study group of 400 cases in which unquestionably a more careful examination of the lungs was made, 14 per cent of the cases showed either pulmonary embolism or infarction—an apparent increase of over 50 per cent.

Classical teaching, as recorded with little variation in the textbooks of two generations, has told us that pulmonary embolism may or may not be accompanied by infarction of the lung. The accepted explanation based on experiment and clinicopathologic correlations of human material is that embolism to a lung with an otherwise normal circulation never produces infarction, whereas an embolus to a lung with a previously impaired circulation, usually by chronic passive congestion, regularly does produce an infarct. Though true in a general way, many exceptions will be met in any series of routine autopsies, many more we believe than is generally recognized. Pertinent figures will be found in the succeeding pages. Moreover, between the two simple categories of embolism without and embolism with infarction we believe a third hitherto unrecognized group of clinical importance must be inserted, embolism with incipient but abortive infarct-like reaction of the pulmonary parenchyma, a group which we have described as incomplete infarction.

In analyzing all of our cases with embolism and infarction—a total of 370 cases—it was found that only 40 per cent were postoperative patients and that of the remaining 60 per cent only half were cardiac patients. In other words, 30 per cent of the cases were noncardiac medical patients, a group in which one does not usually appreciate the danger of pulmonary embolism or infarction.

TABLE I

STATISTICAL DATA ON 370 CASES OF PULMONARY EMBOLISM AND INFARCTION

Postoperative	40% of which 58% had infarcts
Cardiac	30% of which 90% had infarcts
Noncardiac medical	30% of which 62% had infarcts

In a routine autopsy the sources of emboli are usually not searched for too carefully or industriously. Very often restrictions do not permit examination of the extremities. We have observed that a very large percentage of the emboli arise from symptomless thromboses of the deep veins of the legs, usually in the popliteal region. These findings confirm those reported by Rössle,[1] who examined the deep veins of the legs in 400 consecutive autopsies and found thromboses in 27 per cent of the cases over 20 years of age. Of course, in many of the cardiac cases, especially those with rheumatic heart disease and auricular fibrillation, the emboli arise from right auricular mural thrombi. However, even in some of these cases the deep leg veins are also thrombosed.

PULMONARY EMBOLISM WITHOUT INFARCTION

The gross pathology of any organ, and especially the lung, is, of course, the important feature to the roentgenologist, and for the sake of completeness we will review some of the elementary facts. Pulmonary embolism may involve the main branches alone, producing as a rule sudden death, or it may involve some of the terminal branches and be completely asymptomatic. Roentgenograms of acute fatal pulmonary embolism show no definite variation from the normal, but those patients surviving massive embolism may show an acute pleural effusion. Massive pulmonary embolism can occur in either or both main vessels with subsequent organization and superimposed thrombotic extension producing a cor pulmonale. Without a definite history

of an embolic attack, it would be almost impossible to differentiate this condition from a localized thrombosis of the pulmonary vessels. In either of these two conditions the gross pathology consists of dilated pulmonary vessels completely occluded by laminated thrombi. This lesion may exist for years and, because of the very gradually diminishing lumen, enough of a collateral circulation, especially with the bronchial arteries, may be established to prevent infarction.[2] It is of further interest that such lesions very often cause only minimal symptoms. Fragments of this large thrombus, however, may break off and extend farther down the pulmonary tree to produce infarcts.

A case* that exemplifies this condition very well is that of Negress, aged thirty-five, who entered the Massachusetts General Hospital complaining of cough and dyspnea. One and a half years and one year before admission she supposedly had pneumonia associated with fever, cough and rusty sputum. The fever subsided, but the cough continued and was soon accompanied by dyspnea. Figure 4*A* is a roentgenogram taken soon after this admission showing right-sided heart enlargement (cor pulmonale) and dilatation of the hilar vessels. Further evidence for a diagnosis of pulmonary thrombosis was a definite right axis deviation on the electrocardiogram. The patient died very suddenly five weeks later. Figure 4*B* is a photograph of the autopsy specimen showing almost complete occlusion of both main pulmonary arteries by old organized thrombus. It is quite likely that the alleged pneumonic episodes were attacks of pulmonary embolism from which the patient recovered and that secondary thrombosis developed.

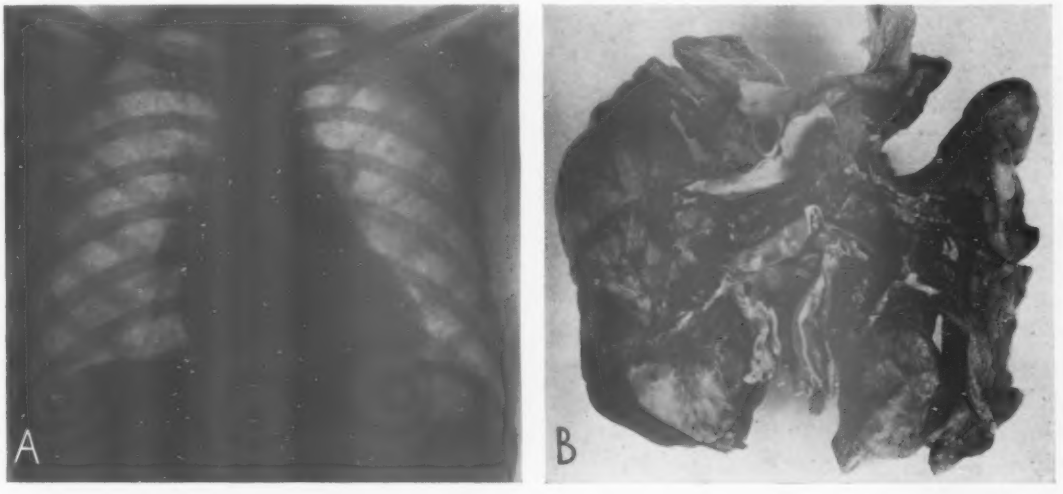

Fig. 4. *A*, case of chronic pulmonary thrombosis. Note enlargement of right heart and hilar vessels. *B*, gross specimen of same case, showing the old organized thrombosis of the main pulmonary arteries.

COMPLETE INFARCTION

Size. Infarcts vary in size from 0.3 to 10 cm., the average being about 3 by 2 by 1 cm., and there are usually several in each lung. A small portion of the margin of the lobe may be involved and such lesions are often no more than 1 cm. in thickness. Such small peripherally placed lesions produce roentgen shadows that have more the appearance of localized pleural thickening than of areas of consolidation.

Location. Infarcts are always peripheral in location, i.e., one or more pleural surfaces are invariably involved. Usually these lesions occur at the junction of two or more pleural surfaces, such as at the costophren-

* This case was reported in detail in the Case Records of the Massachusetts General Hospital, Case 22471, *New England J. Med.*, 1936, *215*, 982–985.

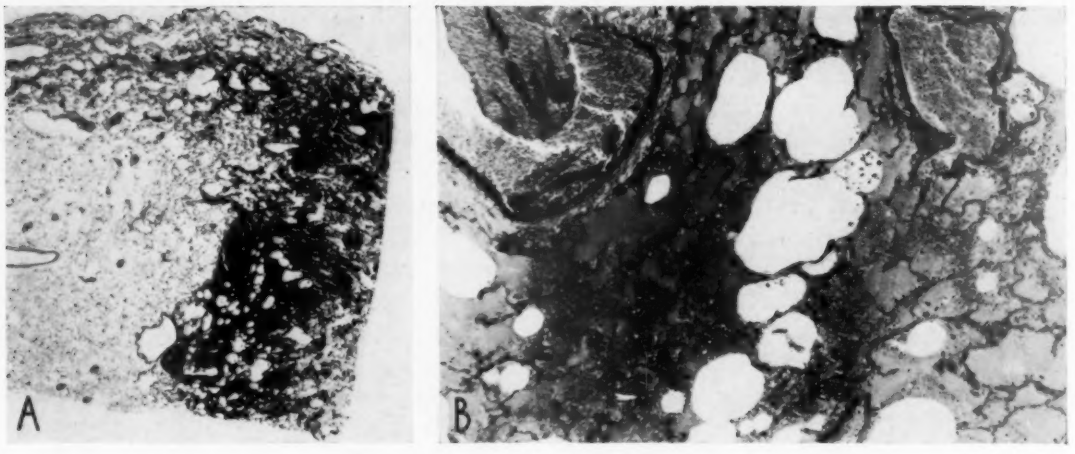

Fig. 5. *A*, photomicrograph of an infarct one day old. Note the absence of a sharp line of demarcation and the presence of air in the atria and alveolar sacs. *B*, higher power photomicrograph showing intact alveolar walls and edema and hemorrhage within the alveoli.

ic angles, at the anterior and posterior lung margins near the mediastinum, or at the margins of the middle lobe. An infarct in the latter location or in a costophrenic angle is very common because the lung is thin and the infarct would be almost completely surrounded by pleura. The distribution of infarcts in our series corresponds fairly well with that recorded in the literature. Seventy-four per cent of them were located in the lower lobes, 43 per cent in the right and 31 per cent in the left. The remainder were distributed almost equally among the other three lobes.

Microscopic Pathology. During the first day and probably the second day of infarction, there is no definite destruction of alveolar walls. There is merely a very marked congestion of the capillaries and diapedesis of the red blood cells into the dilated alveoli of the region involved. The word "infarct" is derived from "*infarcire*," meaning to stuff, and aptly fits this stage of its development. During the first few days only the alveoli, the smallest air-containing unit in the lung, may be filled with blood, while the larger units, such as the alveolar sacs and atria, may still remain aerated. This accounts for the fact that an infarct in this early stage is not uniformly solid grossly

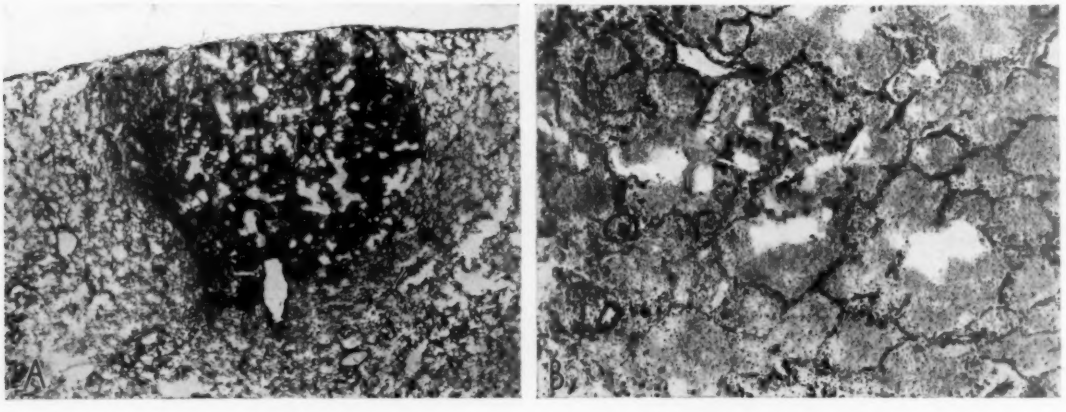

Fig. 6. *A*, photomicrograph of an infarct about four days old. Note its peripheral location, the beginning of encapsulation, and the presence of air in the alveolar sacs and atria. *B*, a higher power showing definite destruction of alveolar walls and more cellular infiltration.

and why its corresponding roentgen shadow may be so difficult to visualize. The roentgen shadow of an early infarct does not usually develop before twelve hours and occasionally not before twenty-four hours after onset of symptoms. During this stage the infarct is often not sharply defined and may simulate focal hemorrhage or bronchopneumonia (Fig. 5).

thrombus or embolus." Because of the onset of the process of organization which usually begins during the second week, the infarct changes from the bright red to the dark or black-red color, becomes more solid more sharply defined and in time even smaller in size (Fig. 6). Depending upon the size of the infarct, organization may last for weeks to months. The granulation tis-

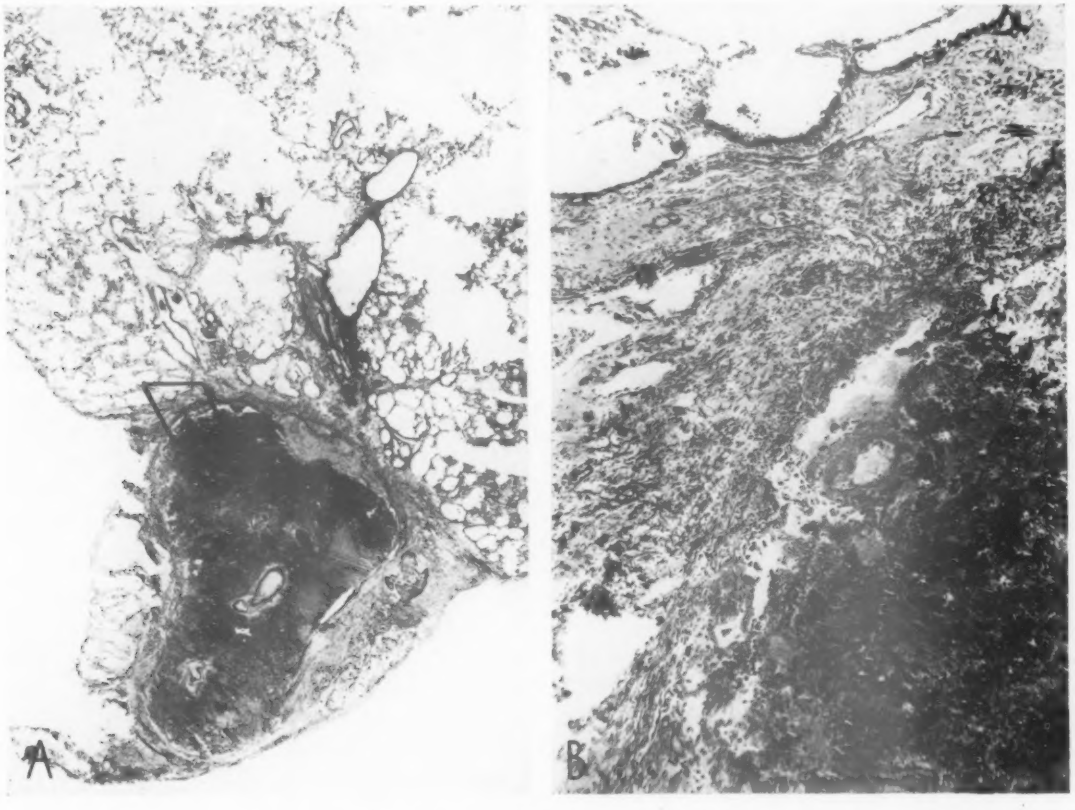

Fig. 7. *A*, photomicrograph of a healing infarct. Note the sharp and thick capsule and the organized and recanalized thrombus in a vessel just proximal to the infarct. *B*, a higher power through the blocked area in *A* showing the thick granulation tissue of the capsule and acellular necrosis within the infarct.

After the second day there is beginning necrosis of the alveolar walls and degeneration of the red blood cells. As this process continues there is dehemoglobinization of the red blood cells, and the appearance of hemosiderin-laden phagocytes and occasionally hematoidin crystals. This stage is in keeping with the usual dictionary definition of the word "infarct"—"necrosis and hemorrhage in an organ, resulting from obstruction of the local circulation by a

sue appears initially at the periphery of the infarct and slowly migrates inward. This marginal organization produces a pseudocapsule which explains the sharp outline and the gradual shrinkage in size of the older infarcts (Fig. 7). A small infarct will become a fibrous scar much sooner than a very large one, the latter often remaining hemorrhagic indefinitely.

Shape. An infarct is sharp in outline and the roentgen shadow cast by it will also be

Fig. 8. Low power photomicrograph of an infarct in a costophrenic showing the "hump"-shaped convexity of its proximal edge.

sharp if the infarct is projected through its greatest thickness. For example, an infarct involving most of a lower lobe will be sharp in outline in the lateral view but indistinct in its upper margin in the posteroanterior view. The shape of the infarct is dependent entirely upon the shape of the part of the lung it involves. Since infarcts are always in contact with visceral pleural surfaces it can readily be seen that these pleural surfaces will form at least one side and more commonly two or three sides of the infarct; thus, to this extent the shape of the lesion must correspond to the shape of the lung involved, and this varies. If the lung tip which fits into the costophrenic angle is infarcted the consolidation may extend for varying distances from the periphery inward toward the heart depending, of course, upon the size of the vessel in which the embolus lodges. Similarly, if a part of the lingula of the left upper lobe or the middle lobe of the right lung is involved, the consolidation extends to the pleural surface or surfaces. If the whole lingula or the whole middle lobe is infarcted, the infarct will of course be the shape of these structures. The long axis of the infarct is always parallel to the longest pleural surface involved. The length and extent of the pleural surfaces involved may vary markedly. For example, the entire lateral and inferior margin of the middle lobe may be involved, producing a long narrow area of consolidation. It is important to note that the cardiac margin of such a lesion has the same general characteristics of a large, more rounded infarct. The medial or cardiac margin of an infarct is convex toward the heart, presents a serrated margin, and, if the central roentgen ray is tangent to it, a a convex, slightly irregular "hump"-shaped shadow is produced. When the lateral costophrenic margins of the lung are involved, the posteroanterior view of the chest shows the "hump"-shaped shadow. When the anterior and posterior costophrenic margins are involved, the "hump" shadow is seen only in the lateral view. Since infarcts may involve any peripheral portion of the lung, including the surfaces of the interlobar fissures, it is often advisable to obtain both oblique and lateral views of the chest before an accurate differentiation can be made between areas of consolidation due to infarction and some other pathology.

While it is true that shadows of infarcts may be roughly triangular in the posteroanterior view of the chest, they are rarely if ever actually triangular in shape. If one studies the margins of the lobes of the lung in their inflated state it can be seen that where two pleural surfaces meet, two sides of a triangle may be formed, but these surfaces are in contact with the chest wall except along the interlobar pleura and therefore the two sides of the triangle formed are not visible, i.e., there is no contrast between the infarct and the chest wall at the points where the triangular shadow is more read-

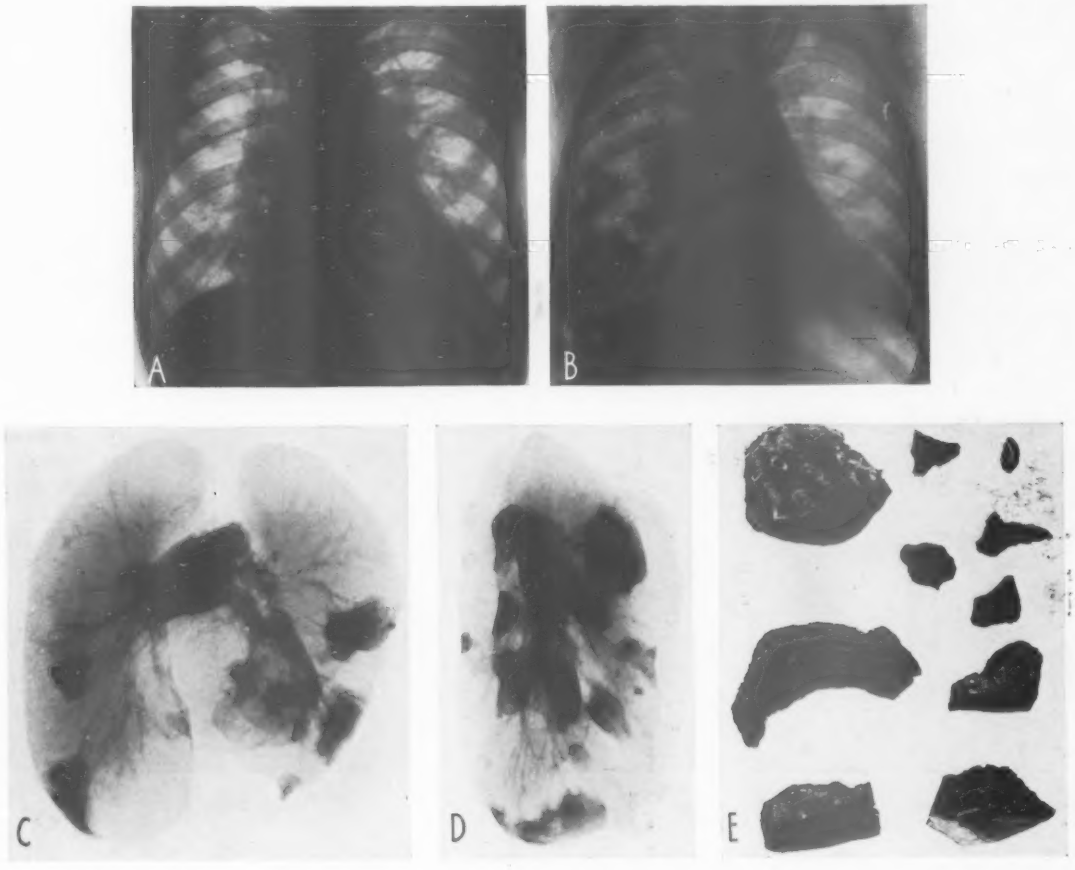

Fig. 9. *A*, patient with rheumatic heart disease, early infarcts at both costophrenic angles and along the right border of the heart, *B*, same subject postmortem, sixteen days later. There are now nine infarcts in the right lung and five in the left, those on the left being partially obstructed by the heart shadow. The triangular densities on the right are due to summation of several infarcts, the interlobar fissure contributing to make a total shadow. The right lung root is partially obscured by a large infarct at the anterior margin of the right upper lobe. *C* and *D*, the lungs of *B* have been removed, injected and fixed with formalin, then inflated and partially dried. The infarcts can be seen more clearly and their true shapes determined. The largest area of density, in the center, represents the infarct in the anterior margin of the right upper lobe. Although the shadows appear triangular in shape in the anteroposterior view, in the lateral view (*D*) they are not at all triangular. The most pointed margin (apex) is directed away from the heart; i.e., to the pleural surfaces. Note that the cardiac margins of all the infarcts are convex instead of pointed. The lesions correspond in shape to the pleural surfaces involved. *E*, ten of the infarcts have been dissected away from the surrounding lung and the irregular shape of the lesions can be better appreciated.

ily visualized. The textbook description of infarcts leads one to believe that they are pyramidal in shape with their apices pointing toward the heart, but this is not true even in instances where a roughly pyramidal or triangular shadow occurs. The apex of the triangle or pyramid is directed away from the heart instead of toward it. The cardiac margin of the infarct is rounded or "hump"-shaped instead of pointed. Figure 8 shows a cross section through an infarct in a costophrenic angle showing the convex "hump" shape at its cardiac border and the sharp apex peripherally.

The size, shape and location of the common infarcts in the lung are well illustrated in Figure 9. Figure 9*A* is an antemortem roentgenogram showing indefinite areas of density along the right margin of the heart and at both costophrenic angles. These shadows represent early infarction but a positive diagnosis could not be made

from this examination alone. Figure 9*B* is a postmortem roentgenogram taken sixteen days later, now showing fourteen separate infarcts in the lung, nine on the right side and five on the left. The largest single infarct occupies the medial anterior margin of the right upper lobe. The lower lateral margin of the upper lobe, the lateral margin of the middle lobe and the lower lateral margin of the left lower lobe also show large infarcts. The triangular shapes of the largest areas of density in the right lung are due to summation of the shadows of several infarcts, the interlobar fissure contributing to make the total shadow. In order to visualize better and demarcate accurately the shape of the infarcts in this case, the lungs were removed at postmortem, fixed in the routine manner by injecting formalin through the trachea, and then dried by blowing air through the trachea for about twenty-four hours under a pressure high enough to keep the lungs distended without forming artificial blebs or rupturing alveoli. Roentgenograms of this specimen (Fig. 9*C* and *D*) show much more clearly the exact shape of the infarcts because the parenchyma adjacent to them is very well aerated. The largest area of density in the center represents the infarct of the anterior

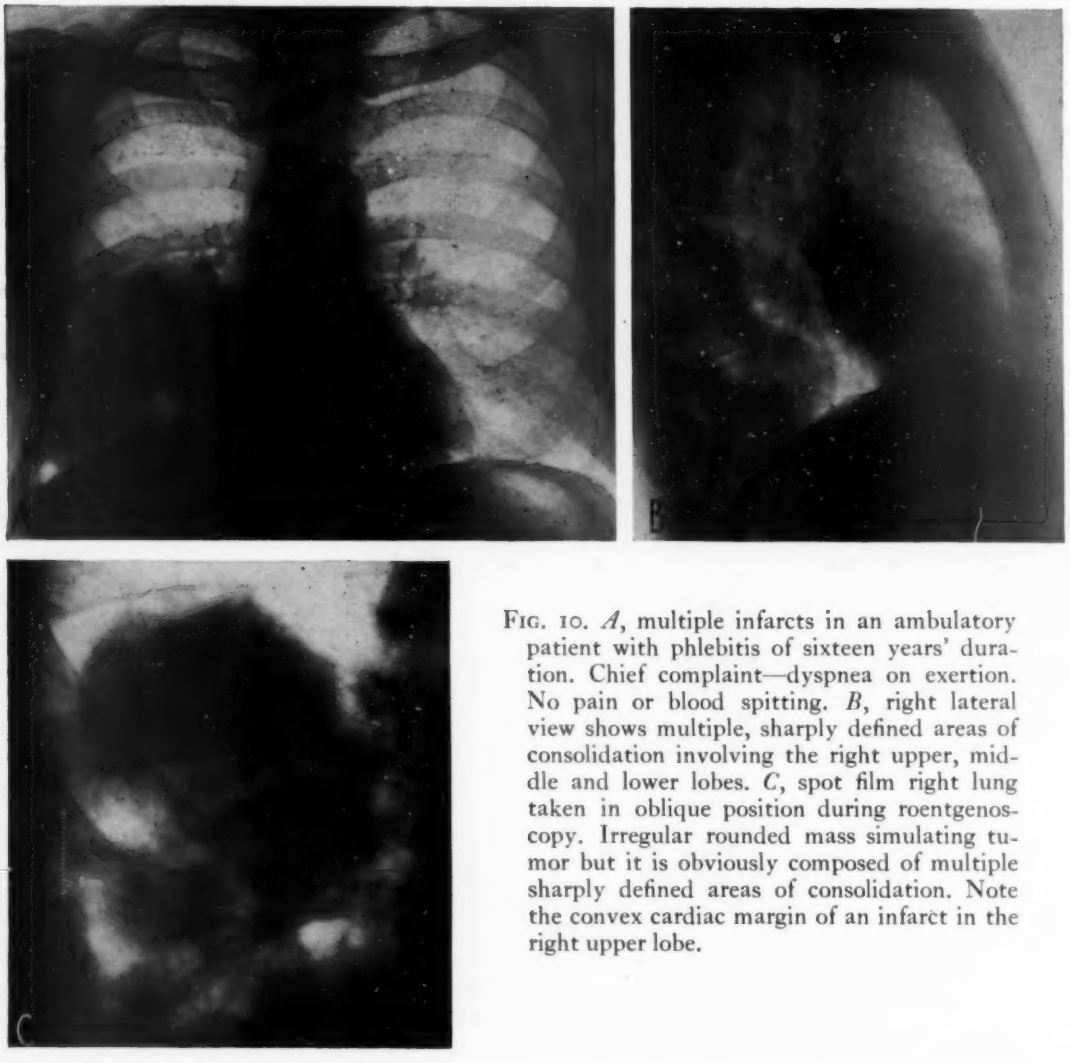

Fig. 10. *A*, multiple infarcts in an ambulatory patient with phlebitis of sixteen years' duration. Chief complaint—dyspnea on exertion. No pain or blood spitting. *B*, right lateral view shows multiple, sharply defined areas of consolidation involving the right upper, middle and lower lobes. *C*, spot film right lung taken in oblique position during roentgenoscopy. Irregular rounded mass simulating tumor but it is obviously composed of multiple sharply defined areas of consolidation. Note the convex cardiac margin of an infarct in the right upper lobe.

margin of the right upper lobe somewhat distorted. This lesion corresponds to the shape of this portion of the lung as do all the other infarcts seen. The medial or cardiac margins of all the infarcts are irregular and convex in shape. The only infarct in the whole group which is truly triangular occupies the right costophrenic angle but the apex of the triangle is directed downward and this triangular shape is not visible on the roentgenograms because there is no contrast between the area of consolidation, the diaphragm and the chest wall. Similar infarcts are seen more clearly in Figures 12 and 16. In summary, it should be noted that none of the infarcts illustrated in Figures 9*A*, *B*, *C* and *D*, are actually pointed toward the heart shadow, that they are not pyramidal in shape, that they are not truly triangular, but that all of them correspond in shape to the portion of the lung they involve and are convex toward the heart shadow. In Figure 9*E*, which shows ten of the infarcts dissected out from the lungs, the irregular shape of the lesions can be better appreciated. Plaster-of-Paris models were made of these infarcts and by the study of these, the characteristics already mentioned are even more obvious.

When more than one infarct occurs in the same lung, and especially when they are closely placed, the sharply defined shadows which vary in shape can be projected so as to produce a summation shadow of nearly any shape. It is therefore very important to attempt to separate the shadows into their component parts and to determine the location, size and shape of these parts. In other words, a "dissection" of a conglomerate shadow for differential diagnosis can be done only by roentgenoscopy and by the taking of roentgenograms in different positions. An example of this aspect of the shape problem is well illustrated in the roentgenograms (Fig. 10) of a fifty-six year old physician who had a history of phlebitis in both legs of sixteen years' duration and who developed pain in the right chest following ligation of several of these veins. The conglomerate dense

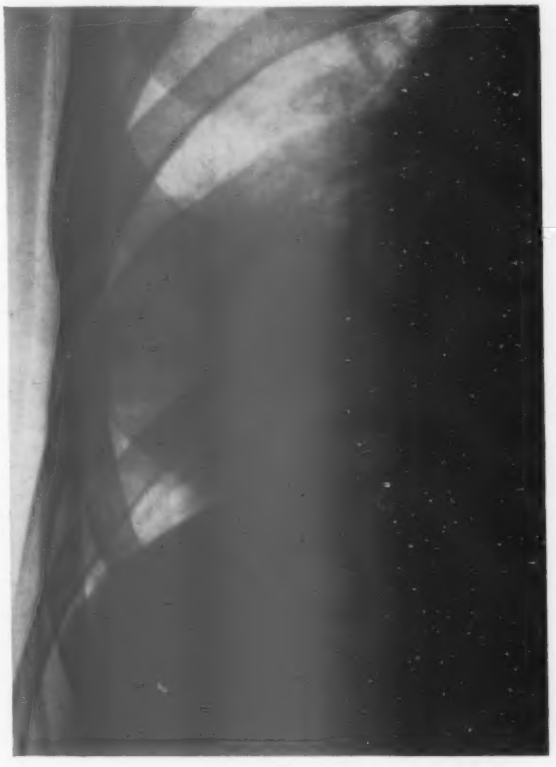

Fig. 11. Postmortem roentgenogram showing two infarcts measuring 5 by 5 by 4 cm. and 3 by 3 by 2 cm., producing, when superimposed in the posteroanterior view, a rounded shadow.

shadows on the right side in Figure 10*A* are seen in the lateral view (Fig. 10*B*) to be composed of sharply defined areas of consolidation arranged along the periphery of the right lower, right upper and middle lobes. They are convex toward the heart shadow, end sharply at the interlobar pleura and the intervening lung is well aerated. In the oblique view taken during roentgenoscopy (Fig. 10*C*), an irregularly rounded shadow is seen, simulating a tumor, but on closer examination it is obviously made up of multiple discrete shadows. The upper margin of the rounded shadow shows the irregular convexity toward the heart, previously described. The lower margin of this shadow ends at a horizontal line corresponding to the base of the right upper lobe. The lower portion of the rounded shadow corresponds to infarction in the middle lobe. During roentgenoscopy the exact location and size of the lesions

could be easily determined. They could be seen to move freely with respiration. The heart shifted to the right on inspiration. A roentgenogram seven months later shows disappearance of the large shadows leaving only dense thin lines.

Round shadows, which one does not usually associate with infarction, may be produced by two or more infarcts if projected in the proper direction. This is seen fairly well in Figure 10*C* in the case described above and a better example is illustrated in Figure 11, a postmortem roentgenogram, which shows a rounded shadow at the right base on the routine posteroanterior view. This shadow in the lateral view is not round but is composed of two infarcts which at autopsy measured 5 by 5 by 4 cm. and 3 by 3 by 2 cm. Although there was aerated lung between the two infarcts, they were so situated, one on the diaphragmatic surface and the other on the anterolateral aspect, that they formed a round roentgen shadow in the posteroanterior view.

Differential Diagnosis. Too often the diagnosis of pulmonary infarction is made only when the clinical symptoms suggest it; i.e., when the patient has chest pain, blood spitting, and shows on examination a pleural friction rub and phlebitis. From our experience, it is quite evident that very often only one symptom and occasionally none is present when an infarct is quite obvious on the roentgenogram. The frequent unwillingness of clinicians to accept a roentgenologic diagnosis of infarct is well illustrated in the case of a man, aged fifty-two, who had a sudden severe right chest pain which lasted for eight hours and which required morphine for relief. One week after the acute attack a roentgenogram (Fig. 12*A* and *C*) showed a rounded shadow at the right costophrenic angle. The roentgenologic diagnosis of infarction was not accepted because there was no history of blood spitting and no clinical evidence of phlebitis. The patient was allowed to proceed about his business until one week

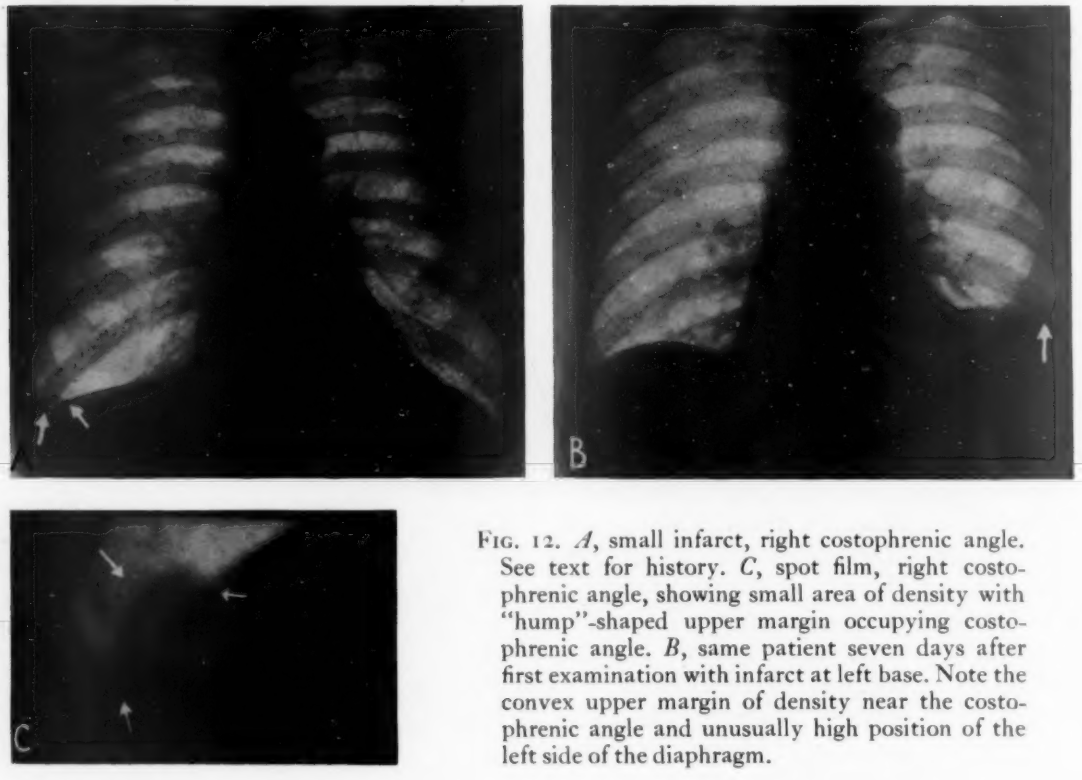

FIG. 12. *A*, small infarct, right costophrenic angle. See text for history. *C*, spot film, right costophrenic angle, showing small area of density with "hump"-shaped upper margin occupying costophrenic angle. *B*, same patient seven days after first examination with infarct at left base. Note the convex upper margin of density near the costophrenic angle and unusually high position of the left side of the diaphragm.

later, when a similar sudden severe pain occurred in the left chest. Another roentgenogram (Fig. 12*B*) showed a similar lesion in the left costophrenic angle. At this time a medical consultant discovered signs of phlebitis in the left popliteal vein and upon more careful questioning elicited a definite history of blood spitting. The patient was kept in bed for two weeks and no further symptoms have developed for over a year.

Most of the clinical signs and symptoms of an infarct are also present in carcinoma of the lung. It is therefore quite important to be able to differentiate the two conditions. In this series there have been 3 cases of pulmonary infarction diagnosed erroneously as cancer, due almost wholly to the fact that an inadequate examination was made. Figure 13 is an antemortem roentgenogram of a patient who was in the hospital several years before this study was begun and whose clinical history was consistent with cancer of the lung. The roentgenogram was interpreted as being consistent with cancer; an infarct was not even considered. The patient died a short time later and at autopsy there was complete infarction of the right middle lobe and the greater portion of the right lower. Certainly the displacement of the heart and mediastinum to the right, the large thickened lung roots and the evidence of reduction in size of the middle and lower lobes suggest malignant disease. It might have been possible, however, to demonstrate small portions of aerated lung between the large infarcts if over-penetrated Potter-Bucky diaphragm roentgenograms had been taken. If normally aerated lung can be demonstrated between multiple, sharply defined, peripherally placed areas of consolidation involving one or more lobes, it follows that the bronchus to the lobe or lobes cannot be obstructed because in bronchial obstruction all of the area supplied by the bronchus should present the same general density. As has already been stated, infarcts reduce the size of the lung, and in this manner may simulate collapse of the lung.

Fig. 13. Antemortem examination showing complete consolidation of the right middle and lower lobes, simulating cancer. At postmortem shadows were due to almost complete infarction of middle and lower lobes.

Fig. 14. Examination twenty hours before death. Infarcts at right base at autopsy: (1) posterior costophrenic angle, measuring 8 by 3 by 9 cm.; (2) anterior inferior margin of the lower lobe near the middle lobe septum, measuring 4 by 3 by 1 cm.; and (3) anterior costophrenic angle, measuring 2 by 1 by 1 cm.; 150 cc. of fluid.

Another example of a case of pulmonary infarction diagnosed erroneously as cancer before admission to the hospital is that of a

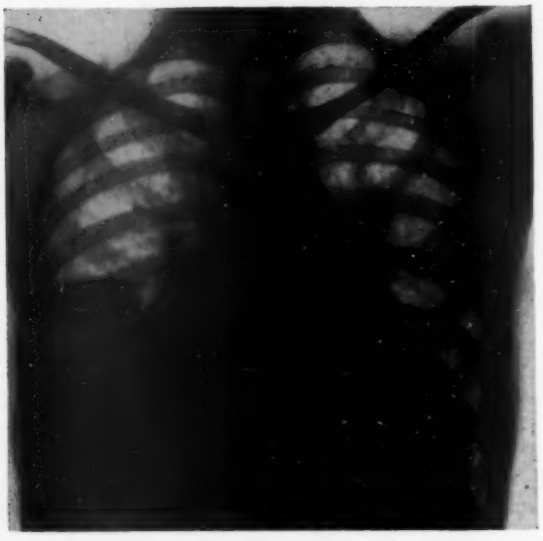

Fig. 15. Infarction of middle lobe simulating interlobar fluid or atelectasis. At postmortem forty days later lesion showed very little change in size or shape.

man, aged sixty, who had pleural pain and blood spitting for five days. There was a slight ecchymosis on the medial aspect of the left knee near a varicose vein. There were no other varicosities and the patient gave no history of injury, pain or discomfort about the knee. The diagnosis of pulmonary infarction was made from the hospital roentgen examination (Fig. 14) but was not accepted by the family physician who proceeded to give the patient exercises for testing his cardiac reserve. About ten minutes after these exercises the patient had an acute pulmonary embolism and died in an hour. Postmortem examination showed that the embolus originated in the left popliteal vein. There were three infarcts in the right lung and 150 cc. of fluid in the right pleural space.

When an infarct involves the right middle lobe, a not uncommon location as has been pointed out in the discussion of the shape of infarcts, the roentgen shadow may be mistaken for atelectasis or interlobar extension of pleural fluid. Figure 15 illustrates just this point. The shadow in the right middle lobe area is sharp in outline, convex inferiorly, and horizontal superiorly, corresponding to the shape of the lateral portion of the middle lobe. It does not extend to the lung root or axillary line. The absence of a fusiform shape differentiates the shadow from pleural effusion. The position of the shadow plus the fact that it does not extend to the lung root differentiates it from middle lobe atelectasis.

That pleural pain may be mistaken for breast pain is well exemplified in the case of a woman who complained of pain in the region of her right breast. Physical examination showed an angioma in this breast. This was removed and twelve days postoperatively she died of an acute massive pulmonary embolism. A roentgenogram (Fig. 16) taken very soon after operation and after another attack of chest pain in the same region as that previously experienced shows a sharp convex "hump"-shaped shadow at the right costophrenic angle quite characteristic of an infarct and another at the inferior margin of the middle lobe. Postmortem examination showed, in addition to the acute embolism, an organizing, definitely old thrombus partially oc-

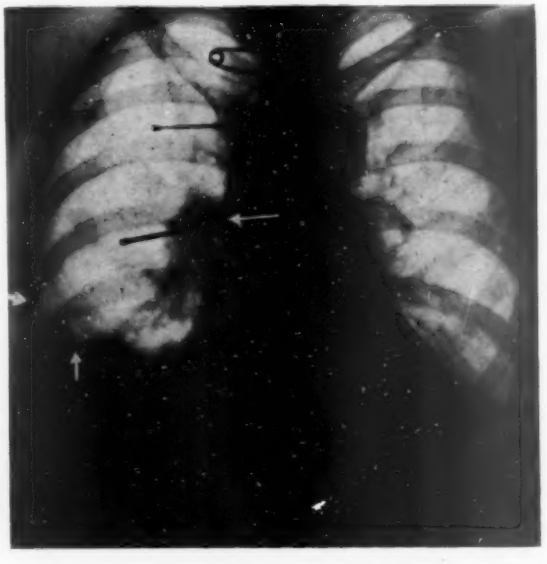

Fig. 16. Area of density in the right costophrenic angle with sharply defined, convex, "hump"-shaped upper border due to infarction. Enlarged right lung root due to chronic thrombosis of the pulmonary artery. Another small infarct is seen at the inferior margin of the right middle lobe, partially obscured by the diaphragm.

cluding a small pulmonary artery leading to a healing infarct at the right base and another more recent one in the middle lobe. There is no doubt that the pain in this case was due to the infarcts, and had a roentgenogram of the chest been taken preoperatively and a diagnosis of infarct been made, the outcome might have been different.

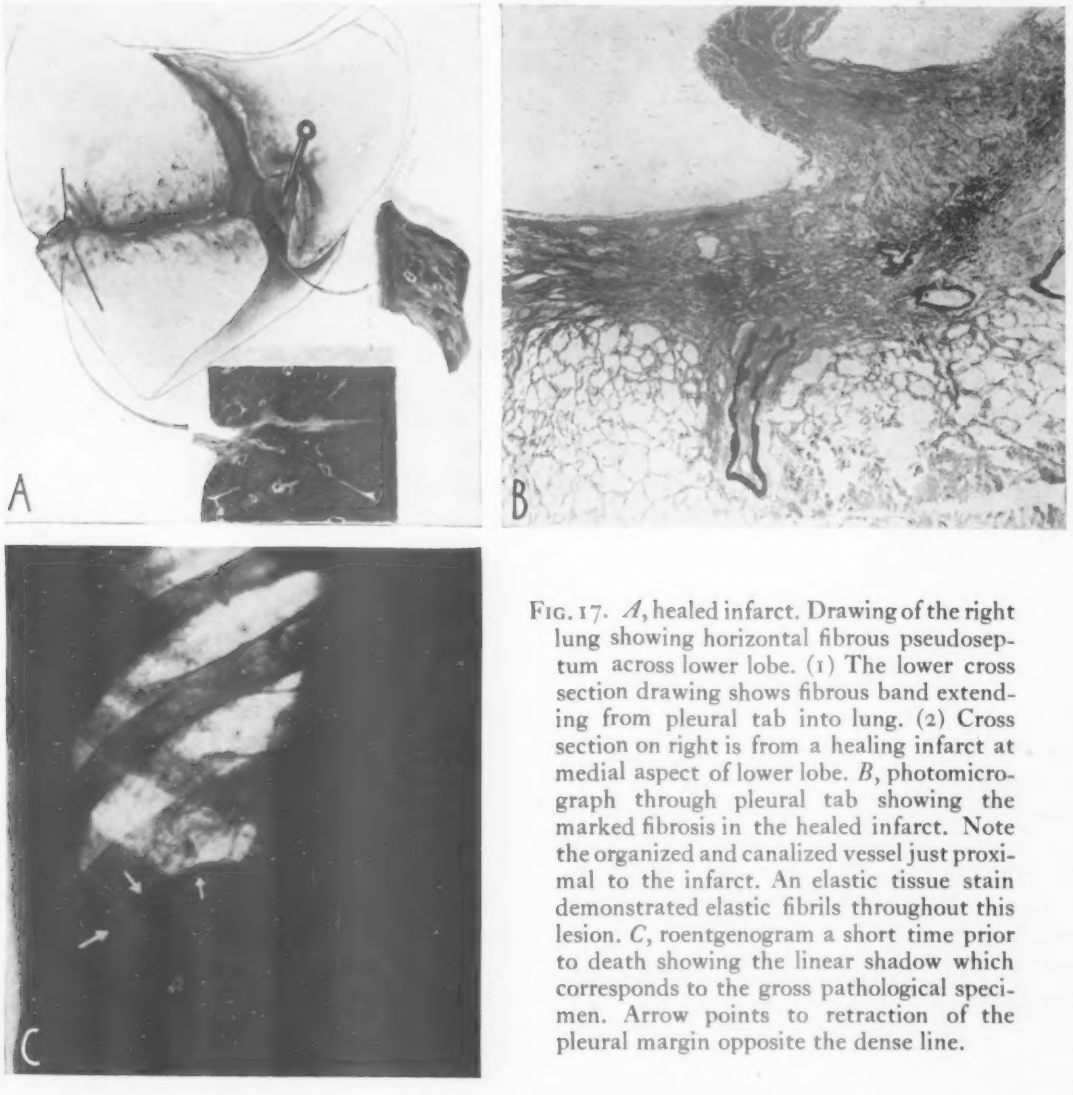

Fig. 17. *A*, healed infarct. Drawing of the right lung showing horizontal fibrous pseudoseptum across lower lobe. (1) The lower cross section drawing shows fibrous band extending from pleural tab into lung. (2) Cross section on right is from a healing infarct at medial aspect of lower lobe. *B*, photomicrograph through pleural tab showing the marked fibrosis in the healed infarct. Note the organized and canalized vessel just proximal to the infarct. An elastic tissue stain demonstrated elastic fibrils throughout this lesion. *C*, roentgenogram a short time prior to death showing the linear shadow which corresponds to the gross pathological specimen. Arrow points to retraction of the pleural margin opposite the dense line.

HEALED INFARCTS AND PLEURITIS

In describing the microscopic pathology of an infarct, we have shown that an infarct usually undergoes a definite cycle, beginning as an acute hemorrhagic lesion, passing through a healing stage, when it becomes smaller, sharper, dense, but maintaining its general shape, and finally ending as an organized healed scar.

Granted that small pulmonary infarcts are not uncommon and that many of them heal, it should be possible to find them at autopsy in their healed stage. It seems improbable that pathologists have systematically searched for the healed infarcts in the past, or possessed any adequate criteria for recognizing them, and consequently the incidence of their occurrence is not known.

This is due to the fact that the usual method of examining the lungs in their fresh deflated state does not permit accurate observations of old scars, pseudosepta, and small linear areas of fibrosis. Our method of examination of the inflated lung with the aid of roentgenograms has made it possible to find these lesions grossly. Once having been demonstrated, they can then be removed for microscopic examination. If the microscopic section shows an old thrombosed or recanalized vessel, we can hypothesize that the lesion may have been an infarct in the past. We have been fortunate in finding a few such cases, in which there was a fairly definite history of an infarct months or years before death.

A good example of a case* showing healed infarction is that of a man, aged sixty-two, who six months before his first entry to the hospital had what was called pneumonia which was followed a few months later by dyspnea and wine-colored pleural fluid. A roentgenogram at the first entry showed fluid at the right base with interlobar extension. A definite parenchymal lesion could not be visualized because of the superimposed fluid. The patient died five months later, about one year after the onset of symptoms. The autopsy showed a linear fibrous band (Fig. 17*A*) running completely across the right lower lobe forming an almost complete septum between the lower and middle thirds. The pleura was thickened over this pseudoseptum and in one place had formed a fibrous tab which was adherent to the parietal pleura. A microscopic section (Fig. 17*B*) across this zone shows masses of closely packed, tangled elastic fibrils—undoubtedly remnants of the original alveolar walls. Two lesions could produce such an end-result, an organized pneumonitis or a healed infarct. In favor of the latter was the finding a short distance toward the hilum from the zone of scarring of a large pulmonary artery which was completely occluded, except for small canalizing vessels, by an organized thrombus. The occluded portion of the artery lay within normal lung tissue, a fact which would be most unlikely if the thrombosis had been secondary to a pneumonitis. We feel confident, therefore, that this lesion was an infarct associated with pleural effusion at the first admission and that it healed completely producing the fibrous band and pleural thickening observed at autopsy. From the drawing of this healed infarct (Fig. 17*A*) it is easy to understand how this lesion could produce a linear horizontal shadow on the roentgenogram.† Figure 17*C* taken a short time before the patient's death still shows some fluid at the right costophrenic angle. The dense oblique line at the right base corresponds to the fibrous band seen in the drawing of the specimen. The retraction of the pleural margin opposite the dense line is visible (see arrow on figure).

Since an infarct of the lung is always peripheral and extends to the visceral pleura, it frequently stimulates a pleural reaction which is believed to be the cause of the pain in this condition. This reaction may or may not give rise to an effusion. In our series of infarcts pleural effusion was present in only 40 per cent of the cases, but in only one-third of this group, or in approximately 13 per cent of the whole group of infarcts, could a causal relation be inferred between infarction and the occurrence of pleural fluid. The fluid in the other cases was more likely due to congestive heart failure or was present on the side that did not have the infarct. Why some infarcts stimulate a serous reaction and others do not is still unsettled. We do not believe that the presence of infection is the provoking agent because secondary infection of an already existing bland infarct is extremely rare. Many infarcts, and especially those that are associated with fluid, will produce pleural thickening when they heal and we believe that if a more careful search is made by both roentgenologist and pathologist, evi-

* This case was reported in the Case Records of the Massachusetts General Hospital, Case 24381, *New England J. Med.*, 1938, *219*, 438–443.

† The differential diagnosis of linear shadows of the lung will be discussed more fully in a later paper.

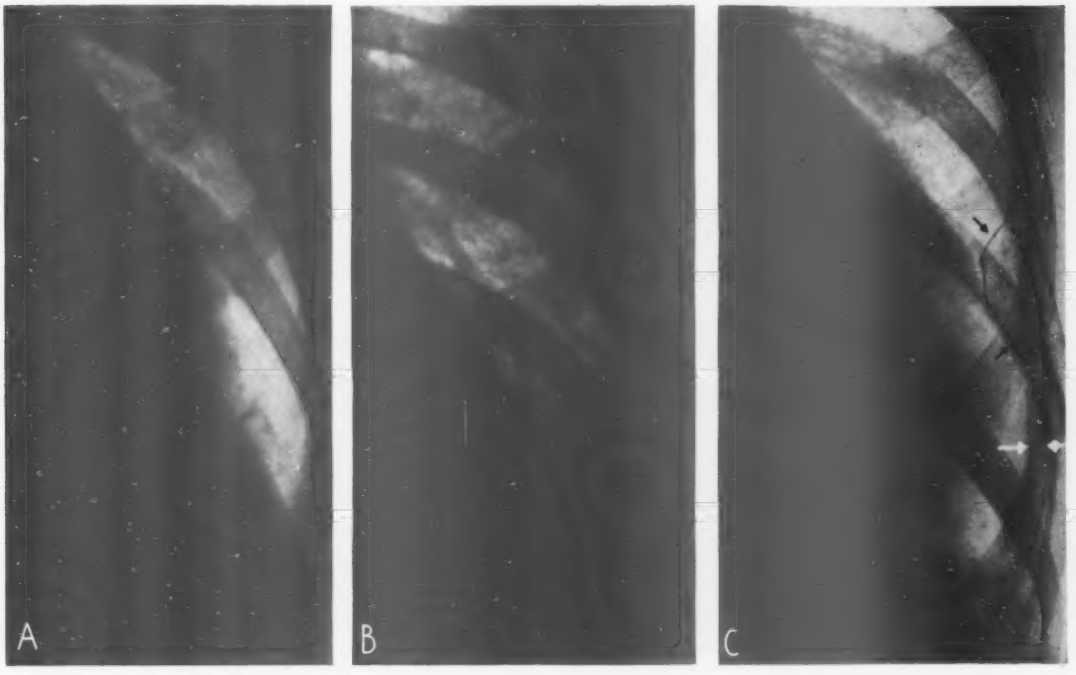

Fig. 18. *A*, normal left costophrenic angle. *B*, symptoms of infarct eight days before; 400 cc. clear fluid. *C*, same patient, one and a half years later. Note the thickened pleura and nodular shadow (retouched).

dence of infarction will be more often discovered as a cause of pleurisy.

While we do not wish to propose that all localized areas of pleuritis, peripheral scars, and anomalous septa are healed infarcts, there certainly appears to be a discrepancy between the very large number of clinical cases of sudden painful pleurisy, many associated with effusion and blood spitting, and the relatively rare pathological diagnosis of healed infarct. This is in part due to the fact that a localized pleuritis is usually assumed to be tuberculous in origin. We believe that if there is no evidence of active tuberculosis in the lungs, and excluding apical adhesions, the presence of a "dry" pleuritis or pleuritis with a sterile effusion is more likely to be due to infarction than tuberculosis. As has already been stated, the absence of a history of phlebitis, surgical operation, or heart disease does not rule out the diagnosis of infarction. If pleurisy with effusion is preceded or followed by a roentgen shadow simulating an infarct, the diagnosis is almost certainly established. Figure 18 illustrates this cycle. The first roentgenogram shows a normal left costophrenic angle in a routine study of a patient with bronchial asthma. The second roentgenogram on the same patient taken eleven days later and eight days after an attack of left chest pain radiating to the shoulder and blood-tinged sputum shows left pleural effusion. Four hundred cubic centimeters of straw-colored fluid were removed. The patient died one and a half years later of coronary thrombosis. The third roentgenogram taken three weeks before death shows chronic pleuritis over the left lower lobe, more marked in the region of the healed infarct at the costophrenic angle and no pleural fluid. These findings were confirmed at autopsy.

INCOMPLETE INFARCTION

Most clinicians and roentgenologists are familiar with the postpartum or postoperative patient who suddenly on about the tenth to fourteenth day develops a little fever, spits up blood, may even have chest pain, and whose roentgenogram shows an infarct-like shadow (Fig. 19*A*). The symp-

toms usually last one to two days and the roentgen shadow disappears completely in two to four days (Fig. 19*B*). This roentgen shadow is usually interpreted as representing pneumonia or an infarct in spite of the fact that, although it is generally believed that infarction cannot occur in a normal lung, there is no evidence of pulmonary congestion or any other abnormal condition. Since most of these patients get well, pathological confirmation of these interpretations has not been made. If this lesion were a true infarct with alveolar wall destruction, it could not disappear in a few days. It would have to heal and organize in the usual way, which has been described above, a process that would take more than a few days. One must infer, therefore, that the lesion did not involve the alveolar walls and that resolution of the process occurred. We have termed this lesion an incomplete infarct, one which terminates by resolution instead of by organization. This same syndrome occurs not infrequently in the ambulatory patient without any indication of a source for an embolus and is sometimes incorrectly diagnosed as tuberculosis.

In 1922 Cutler and Hunt[3] described this clinical and roentgenological syndrome in postoperative patients and stated that the lesions were produced by small emboli arising from thrombi in the operative field. They predicted, probably correctly, that pathologists would be loath to accept these lesions as infarcts. This hesitance may be attributed in part to the questionable relationship between lesions observed clinically in nonfatal cases and those observed both clinically and pathologically in individuals dying of another disease. In addition, although lesions produced by animal experimentations are not always analogous with those occurring spontaneously in man, experimental results in this condition would have been very difficult to evaluate without accurate knowledge of the exact

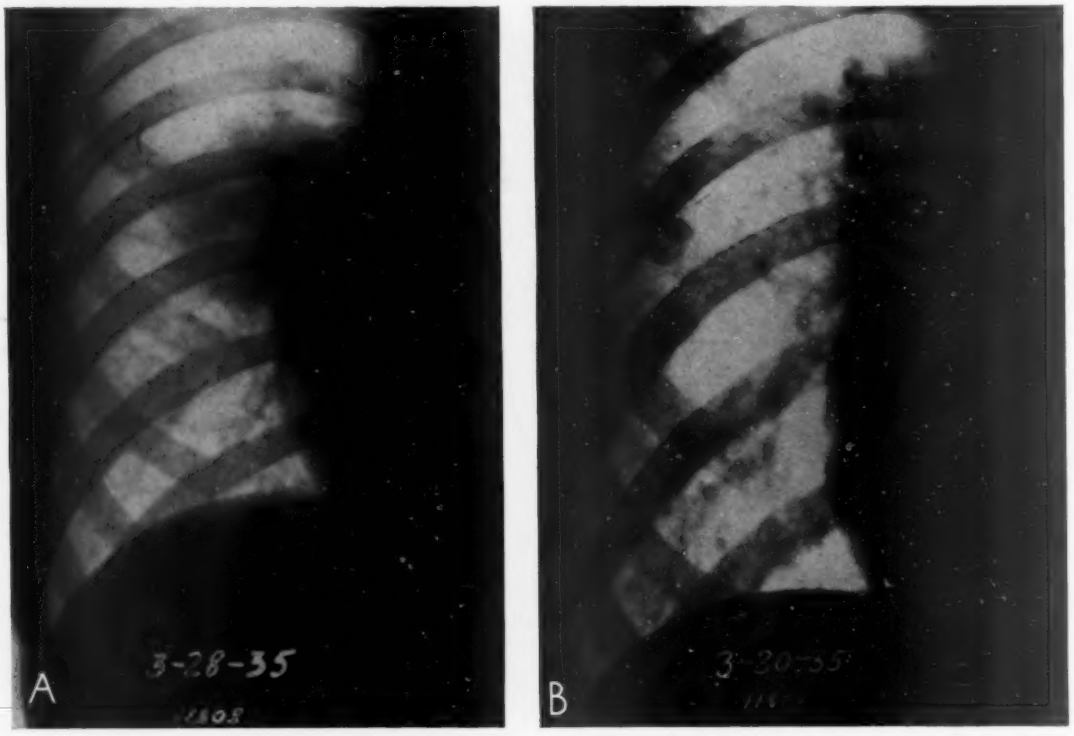

Fig. 19. *A*, incomplete infarct at right border of heart; *B*, same patient two days later. The lesion has disappeared.

morbid anatomy of the human lesion. Practically all the experimental work on pulmonary embolism has shown that a true infarct cannot be produced in a normal lung, i.e., if we accept alveolar wall destruction as the necessary factor in the definition of the word infarct. If, however, pulmonary congestion was produced in the animals before the emboli were introduced, true infarcts developed. Recently Bardin[4] of the University of Paris and Steinberg and Mundy[5] in this country have presented evidence of the production of true infarcts in apparently normal dogs. They both feel that the reason why others have not been able to get comparable results is that they have not used a sufficient number of emboli to produce complete local circulatory obstruction. They neglect the fact, however, that when the pulmonary arteries are loaded with large numbers of emboli experimentally, the circulation is so embarrassed that the lungs are no longer normal. Most of the workers, however, including Cohnheim and Litten,[6] Litten,[7] and Karsner and Ash,[8] did produce lesions in the normal lungs of animals, but because there was no alveolar wall destruction, they apparently ignored their findings. There was, however, edema, red blood cells and a few leukocytes in the alveoli. We repeated these experiments and produced similar lesions* (Fig. 20). We believe that these experimental lesions produced in normal dogs correspond to what we have termed an incomplete infarct in

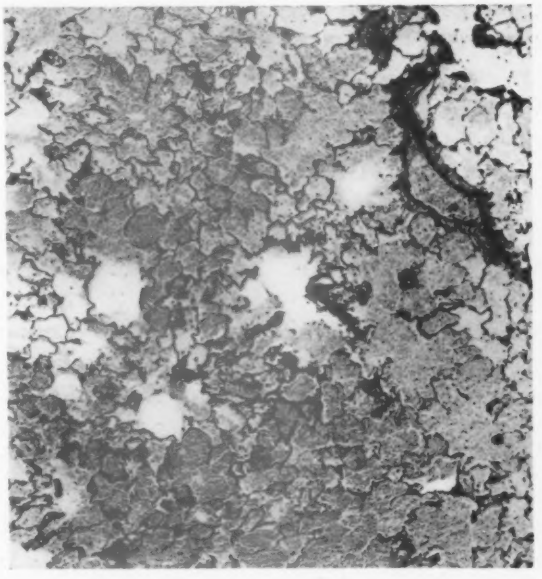

Fig. 20. Photomicrograph of an incomplete infarct produced experimentally in a normal dog. Note the intact alveolar walls and the hemorrhage within the alveoli.

* We wish to thank Dr. John D. Stewart of the Department of Surgery for his help with the animal experiments.

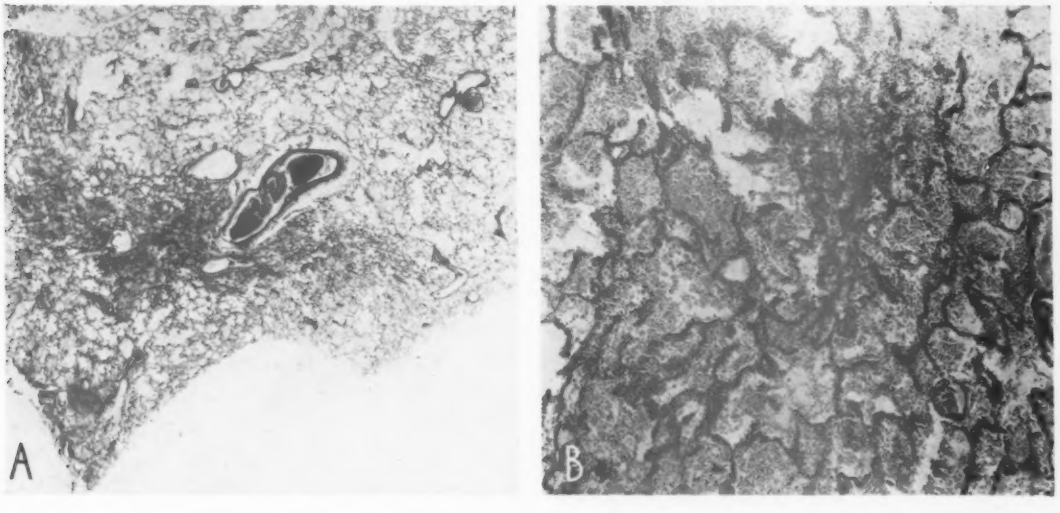

Fig. 21. *A*, low power photomicrograph of an incomplete infarct in a patient who died of acute massive embolism following a pelvic operation. Note the embolus just proximal to the lesion. *B*, a higher power showing intact alveolar walls and hemorrhage and leukocytes within the alveoli.

man. This clinical application of these positive experimental findings, which were previously interpreted as negative results, has not, so far as we know, been made heretofore.

Figure 21 demonstrates an incomplete infarct in a section from the right lower lobe of a woman, aged forty-three, who died of acute massive pulmonary embolism nine days following an operation for uterine suspension. She had complained of a rather indefinite slight right chest pain a few days before her death, but it was so vague that no roentgenograms were taken. The histologic appearance of this infarct is very similar to the lesion produced in the normal lung of a dog, as illustrated above. There is no alveolar wall destruction, merely edema and cellular deposits within the alveoli. The clinical importance of recognizing these disappearing shadows on the roentgenograms, of interpreting them as embolic in origin rather than pneumonic or infectious, and of repeated examinations, cannot be overemphasized, since it may be the only warning signal of a fatal massive embolus. The incomplete infarct is also very similar to the very early stage of a true infarct and it is therefore quite plausible that all these lesions begin the same way, but that the condition of the lung dictates its course—the infarct remaining incomplete in the normal lung and going on to completion when there is disturbance in the collateral circulation, as for example in congestive heart failure.

SUMMARY AND CONCLUSIONS

1. Roentgenological examination of the lungs after death is a definite aid in the pathological examination.

2. A description is given of a routine method of taking postmortem teleroentgenograms and of preparing the lungs by a formalin injection-inflation technique in order to permit an accurate correlation between the anatomic lesions in the lung with those demonstrated on the roentgenogram.

3. From a study of over 3,500 autopsies the incidence of apparent increased frequency of pulmonary embolism and infarction is emphasized and special note is made of the higher proportion among medical rather than surgical patients.

4. The gross and microscopic pathology of pulmonary embolism and infarction is reviewed.

5. The size, shape, usual locations, and the peripheral nature of infarcts are described. Infarcts of the lung are always in contact with pleural surfaces, and the long axis of the infarct is parallel to the longest pleural surface it involves. The cardiac margin of the infarct is convex or "hump" shaped.

6. Special attention is called to healing and healed infarcts, lesions which produce linear shadows on the roentgenograms and which are more easily recognized by the described method of examination.

7. The term "incomplete infarction" is suggested for the syndrome characterized by pleural pain or blood spitting or both, associated with a rapidly appearing and disappearing infarct-like area of consolidation in the lung without alveolar wall destruction. This is shown to be similar to the oft-repeated experimentally produced lesions which had been previously ignored as regards clinical application.

8. Although the correlation of the clinical findings of the cases in this study has not been fully completed, it is apparent that:

a. There is often insufficient clinical evidence of pulmonary infarction to allow a diagnosis; for example, there may be no pleural pain, blood spitting or any obvious source of an embolus, but given any one of these findings, together with roentgenological evidence consistent with an infarct, the diagnosis may be made.

b. In view of the fact that one-third of the cases of pulmonary infarction occur in patients who have not been operated upon and who have no demonstrable cardiac disease, it is important to consider pulmonary infarction in the differential diagnosis of pulmonary disease in ambulatory patients.

c. An accurate diagnosis of pulmonary infarction is very important because unless proper treatment is carried out the patient may have a fatal embolus.

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DISCUSSION

Dr. H. Dabney Kerr, Iowa City, Iowa. It is a pleasure to be able to discuss such a paper as has been presented by Drs. Castleman and Hampton. We always expect something good to come out of the Massachusetts General Hospital and we certainly have not been disappointed this time.

There has been very little theorizing in the presentation of this excellent work, and we have been given a maximum of factual evidence, which, it seems to me, is one of the things which we as roentgenologists need most. Certainly all of us have been puzzled by various shadows seen in the antemortem films and even in the postmortem films of inflated lungs which we may have taken, but it seems to me that the work which has just been presented is a tremendous help to us because it shows us basically the causes of these shadows over which we may have theorized but which we were unable to explain adequately.

Roentgenologists need this type of basic, fundamental pathologic and anatomic verification of the shadows which they see on films. I can plainly see how this can be an aid to us. It gives us a much better insight into the pathologic processes with which we are concerned and which we commonly encounter. It is also an example to us, those of us who may think we are terrifically busy and thereby put aside the basic explanation of roentgenologic findings, because certainly if anyone is busy it is the group at the Massachusetts General Hospital. Yet they have taken time to give us this exceedingly important and clarifying paper.

Dr. Castleman has told me that at least 30 per cent of their cases of proved infarction and embolism have occurred in noncardiac medical patients. That, to me, was very illuminating because it is not usually thought that noncardiac medical patients and nonsurgical patients have these infarcts.

It has been interesting to me also to notice that the shape of these lesions is not always the so-called triangular shadow; that the position of the infarct, particularly in the majority of cases where they are found at the bases, is not that of the triangle with the base to the periphery but is more likely to show either an oval or a triangular shadow with the base medial and the apex peripheral. They have given us both in the pathologic specimens and on the films very adequate explanation for these findings.

I think this is undoubtedly one of the best papers giving us a fundamental explanation of the pathological findings in the chest that we have had the pleasure and opportunity of hearing for a long time.

Dr. George W. Holmes, Boston. I would like to add a brief statement as to the practical value of this method of examining the chest; perhaps I should not say "add," but "emphasize."

First, most of these patients are too sick to be brought to the roentgen department for examination and that means that the examination must be made at the bedside. We need better portable apparatus than we are usually equipped with to do the best work. To do our best work, we should be able to take a film at the bedside in one-twentieth of a second. Such apparatus is now available in some clinics.

The second point which I would like to make is that a single examination, as Dr. Hampton

pointed out, may be negative and we should be given the opportunity to examine these patients more than once and on different days.

Then, I would like to emphasize again the peculiar character of these shadows. They are not what we expect to see.

I want to thank Dr. Kerr personally for emphasizing in his discussion the amount of work that these men have done.

DR. DAVID S. DANN, Kansas City, Mo. I would like to make one or two comments that occurred to me.

Of course, all of us here undoubtedly feel, as I do, the great value of this fundamental piece of work and feel the justification for the careful correlation between the pathologic and the roentgenologic departments, and undoubtedly that will explain away many embarrassing moments that most of us have had at one time or another.

I have been very much interested in the statement concerning the emboli occurring postpartum; that is, noncardiac in nature, and it occurs to me now, as I recall seeing sections presented by our pathologist in which he demonstrated emboli of placental cells occurring normally, I wonder whether these emboli which have been demonstrated in this fashion may not represent such embolic phenomena.

DR. CASTLEMAN (closing). In answer to Dr. Dann's question about placental cells, I must admit that I have not heard of it. However, the occurrence of incomplete infarction in postoperative patients as well as the postpartum would seem to indicate that embolic placental cells, if true, is not an adequate explanation.

Vol. 43, No. 3

CYSTIC BRONCHIECTASIS*

A CLINICAL AND ROENTGENOLOGICAL STUDY

By DAVID REISNER, M.D., *and* I. G. TCHERTKOFF, M.D.
NEW YORK CITY

CYSTIC disease of the lung has attracted considerable attention in recent years as indicated by the rapidly accumulating literature on this subject. There is no doubt that cases of pulmonary cystic lesions are being observed with increasing frequency. In a large measure this is probably due to the fact that this condition is at present more readily recognized than heretofore. Another reason, however, may be the increasingly wide use of routine roentgenographic examinations of the chest, both in general hospital practice and in apparently healthy individuals. In a number of instances pulmonary abnormalities that may otherwise have remained undetected are thus brought to light.

It is not the purpose of this paper to enter into a full discussion of the various forms of air-containing cystic changes in the lung, but to call attention to a particular type of lesion, examples of which are often included in reports on cystic pulmonary disease. We refer to cases which have been variously designated as "polycystic lung" (Bagliani[3]), "bronchiectasis universalis cystica" (Dániel and Jezsovics[8]), or "open honeycombed lung" (*offene Wabenlunge*, Lenk[19]). From a purely morphological point of view it may be said that pulmonary changes of this type occupy a somewhat border line position between cystic formations and saccular bronchiectasis inasmuch as they combine certain characteristic features of both. However, our observations would tend to indicate that this lesion represents a well defined entity characterized by distinct clinical and roentgenographic features as well as by corresponding pathological changes. It therefore seems justified to separate cases of this type, on the one hand, from other forms of cystic disease of the lung and, on the other, from common bronchiectatic lesions resulting from various inflammatory processes. In addition, it is of particular practical importance to differentiate such lesions from pulmonary tuberculosis as will be brought out in the subsequent discussion.

This report is based on a group of 17 cases which were observed on the Tuberculosis Services of the Metropolitan and Sea View Hospitals and in the Tuberculosis Clinics of the Department of Health. While the total number of cases studied was considerably larger some were eliminated from this report, either because of insufficient data or because of complicating features which rendered the identification of the original character of the lesion rather difficult.

CLINICAL ASPECTS

Of the 17 cases, 12 were males and 5 females. Sixteen were white and 1 was colored. The ages ranged from eleven to seventy years. There were 3 patients, eleven, thirteen and fourteen years old respectively at the time of the first observation. The age factor as found in this series of cases cannot be regarded as of any particular significance. The fact that the majority of the individuals were adults is explained by the general nature of the material at our disposal.

History and Symptoms. In a large proportion of the cases symptoms attributable to the respiratory tract were either entirely absent or very mild. This was often quite out of proportion with the marked physical signs and, even more so, with the extent of the pulmonary changes as demonstrated on roentgen studies. In a number of instances the pulmonary condition was discovered on

* From the Sea View Hospital, Staten Island, N. Y., the Tuberculosis Division of the Metropolitan Hospital, and the Clinics of the Bureau of Tuberculosis of the Department of Health, New York City.

routine roentgenograms. This was the case in 7 patients of this group.

Of the 17 cases, 4 had practically no complaints referable to the respiratory organs. In 8 patients the symptoms were quite mild, such as recurrent "chest colds" or "bronchitis," slight chronic cough of many years' duration, either dry or associated with expectoration of scanty sputum. The remaining 5 cases presented more definite symptoms, such as more or less severe cough, usually exceedingly chronic, with or without expectoration. In some cases the symptoms appeared to be subject to seasonal exacerbations, usually during the winter months. In none of the cases was the expectoration fetid in character. Two patients gave a history of a single hempotysis during the entire course of illness of many years' duration. In one of these there was evidence of a cardiovascular complication with signs of congestive failure. Several patients complained of progressive shortness of breath on exertion. This was observed in individuals of advanced age with pulmonary emphysema or cardiovascular complications. Constitutional signs and symptoms, such as fever or loss of weight and strength, were absent in the uncomplicated cases.

Aside from the usual childhood diseases, the past histories did not contain any significant data, especially as regards preceding respiratory affections. Only 2 patients had a history of pneumonia in the past. While the actual date of onset of the symptoms and their total duration were extremely difficult to determine because of the mild and rather vague character of the complaints, it is noteworthy that a number of cases dated the onset of their symptoms to early childhood. In others, a history of twenty years' duration or more could be obtained.

Physical Examination. In the absence of complications the general condition shows little, if any, impairment. This is, of course, different in cases showing signs of either chronic respiratory infection, emphysema or circulatory embarrassment. Varying degrees of dyspnea and cyanosis were found in several of the older individuals, evidently due to coexisting emphysema or cardiovascular complications. Clubbing of fingers was found in 3 cases, all of whom showed evidence of either chronic pulmonary infection or congestive cardiac failure.

Examination of the chest as a rule shows no visible deformity or retraction of the thoracic wall. The percussion note over the involved area is variable, that is, either impaired, or of normal resonance, or else hyperresonant, depending upon the extent of involvement and the coexisting emphysema. The breath sounds are either bronchovesicular or bronchial, but at times diminished in intensity. Almost invariably one finds numerous moist râles of medium or coarse size and many rhonchi. Since these signs are often particularly pronounced over the upper lobes, it is quite understandable that an extensive tuberculous process is frequently suspected in these cases.

Laboratory Findings. The usual laboratory examinations did not disclose any relevant findings. The sputum was consistently negative for tubercle bacilli in all but one case in which the roentgen findings indicated the presence of a complicating tuberculous lesion. The absence of tubercle bacilli from the sputum merits particular emphasis in view of the fact that the majority of the cases of this series was referred for institutional care with the diagnosis of pulmonary tuberculosis. A number of the cases had been in tuberculosis hospitals or under clinic supervision over a period of many years. The blood Wassermann test was positive in 2 cases. Although lues has been referred to by some authors (Sandoz,[24] Balzer and Grandhomme[4]) as a possible etiologic factor in this type of pulmonary lesion, it seems reasonable to regard the presence of a luetic infection as a mere coincidental finding.

ROENTGEN FINDINGS

Roentgen examination provides most pertinent information as to the location of

the lesion, its distribution and, above all, an accurate portrayal of the morphologic character of the changes.

The extent of involvement varies greatly in the individual cases. In most cases, however, the lesion occupies rather large areas of the lung, such as the greater portion of one lobe, a whole lobe, or even several lobes. Table I indicates the distribution and the extent of involvement as found in this series. It will be noted that there was a marked predominance of cases in which the right lung was involved. Another noteworthy point is the frequent involvement of the upper lobe. This deserves particular mention as it may be of value in distinguishing lesions of this type from ordinary saccular bronchiectases which, as is generally known, are more commonly situated in the basal portions of the lung. On the other hand, it is well to remember that the frequent location in the upper lobes may cause difficulties in differentiating such changes from tuberculous lesions.

The characteristic roentgenographic findings are as follows: The involved portion of the lung shows a honeycomb- or sponge-like appearance and often gives the impression of a bizarre network composed of meshes of various sizes. This appearance is produced by a cluster of thin-walled, closely packed cavities separated by fine trabeculations. The cavities are usually round or ovoid, at times their borders are polygonal or irregular. This bizarre structure is evidently due to overlapping of the borders of the cavities located in various planes of the involved pulmonic area. The size of the cavitations is variable, usually ranging from that of a pea to that of a walnut. Small fluid levels are often noted at their lower poles especially in those situated in the more dependent portions of the lung. Changes indicative of a chronic inflammatory process, such as marked fibrosis or shrinkage of the lung, are as a rule absent in uncomplicated cases of cystic bronchiectasis. In most cases there is no evidence of alteration in the position or shape of the mediastinal structures or heart, no narrowing of the intercostal spaces and no distortion of the contours of the thorax. The aeration of the involved portions of the lung shows little impairment and its volume does not appear appreciably reduced although the lesion may be quite extensive.

The roentgenographic findings just described represent the basic and characteristic pattern of the lesion. Some cases, however, show more or less pronounced deviations from the typical appearance, chiefly varying degrees of surrounding fibrosis and infiltration with resulting retraction of the pulmonic field. Such additional changes are indicative of a superimposed chronic inflammatory process. The typical picture of the lesion is generally clearly preserved in young individuals. Complicating features are more apt to be found in older persons, especially in cases presenting marked subjective symptoms and a history of recurrent respiratory infections of many years' duration.

Table I

DISTRIBUTION OF THE LESION

	No. of cases
Right upper lobe	6
Right middle lobe	1
Right upper and middle lobes	2
Right middle and lower lobes	1
Right upper, middle and lower lobes	2
Left upper lobe	1
Massive involvement of total left lung	2
Bilateral involvement	2
Total	17

A type of lesion which may represent a somewhat different entity was found in 2 cases included in this report (Cases XVI and XVII). While the clinical picture of these cases seemed to conform with the one observed in the other cases of this group, the roentgen findings were somewhat different, inasmuch as there was marked contraction of the pulmonic field accompanied by displacement of the mediastinal structures and heart. Massive involvement of an entire lung with complete absence of functioning parenchyma may account for this

roentgenographic picture. A case in which the roentgen findings were practically identical with those just mentioned was recently reported by Gale, Keeley and Coon[13] following pneumonectomy.

Bronchographic examinations were carried out in 9 cases of this series. In nearly all of these cases the contrast material readily entered and filled the above described cavities. This demonstrated that they represented saccular dilatations communicating freely with the bronchial tree. Some of the cases also showed cylindrical and fusiform bronchiectasis leading directly into the dilated sacs. There were, however, no indications of distortion or irregularity in the shape or course of the saccular or cylindrical dilatations, as contrasted with ordinary bronchiectasis of inflammatory origin where such findings are usually present.

PATHOLOGY AND PATHOGENESIS

A perusal of the literature disclosed only few reports on the pathological findings in cases which clinically and roentgenographically were comparable to those discussed in this paper. Such instances were reported by Herms and Mumme,[16] Weiss,[28] Canigiani,[6] Nolte,[20] and quite recently by Sellors.[26] A similar case is mentioned by Aschoff.[2] One of our cases (Case XIV) came to necropsy and the findings are included in the case report.

The gross findings are as follows: A large portion of the lung, usually one or more lobes, is transformed into a cluster of thin-walled, cyst-like cavities of variable sizes which communicate freely with the bronchial branches and sometimes with each other. These changes give the involved portion of the lung a characteristic sponge-like appearance. The cavities have a smooth and shiny inner surface and frequently contain small amounts of mucoid secretion. In addition to the cyst-like sacs there are also fusiform and varicose dilatations of the larger bronchial branches which show a regular even lumen and often lead directly into the saccular dilatations. The pulmonary parenchyma is either absent or considerably reduced, the remaining alveoli showing emphysematous changes. The yellowish-white color of the involved area, due to marked diminution or absence of anthracotic pigment, contrasts with the dark discoloration of the adjacent normal portions of the lung.

Microscopic examination reveals that the walls of the cystic dilatations contain the structural components of the bronchial walls, namely, muscle, cartilage, mucous glands and epithelial lining. The amount of cartilage and muscle shows considerable variation, from complete absence in some cases to irregular proliferations in others. The epithelial lining is of a true bronchial type and consists either of several layers of cylindrical ciliated cells, or of a single layer of either cylindrical, cuboidal or flat cells (Sellors). The septa between the sacculations consist of a thin layer of loose or compact connective tissue. At times there is an excessive amount of fibrous tissue between the cavities. Some cases, however, show, in addition to the above findings, varying degrees of subacute and chronic inflammatory changes with more or less extensive bronchitis and peribronchitis. Such changes are evidently the result of superimposed infection which is bound to complicate the original condition and cause a marked alteration of the typical anatomic findings.

While the anatomic and morphologic character of the pulmonary changes just described appears quite well defined, the question as to the pathogenesis cannot be regarded as having been satisfactorily answered as yet. In considering the pathogenetic aspect of cystic bronchiectasis, it seems necessary to refer briefly to the pathogenesis of cystic disease of the lung in general.*

One type of pulmonary cystic lesion has been considered as due to a congenital neoplasia and designated as "fetal cystic bronchial adenoma" or "cystadenoma"

* It should be mentioned in this connection that only air-containing cystic lesions of the lung are considered in this paper.

(Stoerk,[27] Couvelaire[7]). In these cases the larger bronchi were found to be normally formed, while the lung parenchyma appeared to be replaced by numerous small cysts and a stroma containing gland-like epithelial proliferations. It is obvious that there is no similarity between this form of cystic disease and the type referred to in this report as cystic bronchiectasis.

Some investigators postulated intrauterine inflammatory processes resulting in stenosis of the bronchi as a cause of cystic changes in the lung (Grawitz,[14] Stoerk[27]). It is quite probable that bronchial stenosis plays a certain rôle in the formation of the large sac-like or "balloon" type of cysts which are often connected with the bronchial tree by a very narrow opening (Dehio,[9] Pollock and Marvin,[22] Anspach and Wolman,[1] and others). This mechanism, however, could hardly account for the formation of the cystic variety of bronchiectasis since the latter show no evidence of bronchial stenosis. In fact, the presence of a wide open communication with the bronchial tree must be regarded as one of the typical features of this condition.

Other observers, especially Heller,[15] thought that fetal atelectasis with consecutive hyperplasia and dilatation of the bronchi might be responsible for the origin of the "sponge-lung" or cystic type of bronchiectasis. This assumption, however, fails to explain the fact that such changes have been found in the newborn.

The majority of the writers on the subject are inclined to regard as the most important cause of cystic lesions of the lung an arrested development of the bronchial tree combined with agenesia of the pulmonary parenchyma (Hueter,[18] Hückel,[17] de Lange,[10] Schenck,[25] Sellors,[26] and others).

It may not be amiss to recall here some of the essential facts concerning the embryonal development of the lungs and bronchi. It is well known that the bronchial tree develops from successive dichotomous subdivisions of the trachea and stem-bronchi. Pulmonary alveoli appear as small lateral sacculations of the terminal bronchioli during the fifth and sixth months of intrauterine life. If the normal subdivision of the bronchial tree is impeded at a certain phase of the embryonal development, no lung parenchyma will be formed and the already existing bronchial ramifications may continue to grow into abnormally large spaces. Following the inflation of the lungs after birth these spaces will become distended and form cystic air sacs.

It is conceivable that the variations in the types of pulmonary cystic formations, especially as to size and number, depend to a great extent upon the stage of intra-uterine life at which the arrest in the development of the bronchopulmonary structure occurs. If its growth is impeded in the early period of embryonal development, at a time when only the stem-bronchus or the lobar branches have been formed, the parenchyma of an entire lung or of a lobe will be absent and the resultant malformation may be one or several large cystic sacs. This may be the mechanism for the formation of large solitary or multiple cysts, most of which are encountered in infants and in young children (Anspach and Wolman,[1] Ribadeau-Dumas and Rault,[23] Eloesser,[12]) on rare occasions only in adults (Dehio,[9] Wood[29]). If, on the other hand, the development is arrested late in fetal life, that is after the evolution of the bronchial tree has progressed to the stage of subdivision into small or terminal bronchioli, but before the formation of the pulmonary alveoli, the result may be an ectasia of numerous small bronchioli, a condition sometimes described as diffuse bronchiolectatic emphysema.

The cystic form of bronchiectasis, which is the subject of the present report, may be said to occupy an intermediary position between the large cystic sacs and the bronchiolectatic cystic disease. An impeded development of the bronchial tree occurring after the large and the medium sized branches have been formed, which prevents their further subdivision into smaller ramifications, may explain the origin of this type of cystic dilatations of the bronchi.

It should be pointed out, however, that

the pathogenesis of cystic disease of the lung in general and of the cystic form of bronchiectasis in particular is not completely understood as yet, since so much of it is purely conjectural. It is quite conceivable that the resultant changes represent the combined effect of more than one of the above mentioned causes. A combination of an impeded development with a defective supporting stroma and insufficiently formed elastic elements, as emphasized by Pepere[21] and Bard,[5] must also be considered as one of the possibilities. Furthermore, it should be mentioned that it is not always possible to draw a sharp line of division between the individual types of cystic formations and that some cases may combine characteristic features of two different groups (Dustin;[11] see Case XVI of this series).

It must be admitted that direct and unequivocal proof of the congenital origin of cystic bronchiectasis is extremely difficult to obtain, even in the presence of necropsy findings, unless observed in a very young infant. This, however, is rarely the case as this condition is seldom, if ever, the direct cause of death in that age. In older individuals, on the other hand, superimposed chronic inflammatory changes may often obscure the original character of the lesion. The following pathologico-anatomic features, however, may be regarded as important criteria in favor of the congenital nature: the lack of anthracotic pigmentation in the involved portion of the lung; the regularity in shape and distribution of the saccular dilatations; their wide open communication with the bronchial tree; the frequent predilection for the cranial portions of the lung; and the absence of marked inflammatory changes, in contrast to the cases of ordinary bronchiectasis of a comparable extent.

A number of clinical indications lend support to the concept of the congenital origin, namely: the disproportion between the frequent asymptomatic or extremely mild clinical course on the one hand, and the extent of the pulmonary changes on the other; the insignificant past history, especially as regards past respiratory infections; and the fact that in many cases the onset of the symptoms may be traced back to early childhood. To this must be added the characteristic roentgen findings which are strikingly different from the changes observed in the common forms of bronchiectasis.

DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of cystic bronchiectasis the following conditions deserve particular consideration: saccular bronchiectasis resulting from chronic inflammatory processes; tuberculosis; emphysematous bullae; and other forms of cystic disease of the lung.

(1) *Saccular Bronchiectasis.* The clinical and roentgenological distinction between ordinary saccular bronchiectasis and the honeycomb- or sponge-lung characteristic of cystic bronchiectasis was repeatedly stressed in the foregoing paragraphs and needs no further elaboration at this point. It should be remembered, however, that in some cases a differentiation from ordinary saccular bronchiectasis may be extremely difficult because of superimposed chronic infection. In fact, it is entirely possible that a number of cases with the usual clinical and roentgenographic manifestations of chronic suppurative bronchiectasis represent the end-result of an infectious process engrafted on a pre-existing honeycomb lung.

(2) *Tuberculosis.* The physical signs frequently found in cases of cystic bronchiectasis, such as altered breath sounds and numerous moist râles over the upper lobes, very often suggest an erroneous diagnosis of pulmonary tuberculosis. The scant subjective symptoms, the good general condition of the patient and the non-progressive clinical course, present a striking contrast to the physical signs and are important points against tuberculosis. Roentgenologically, the differentiation from secondary bronchiectasis due to chronic fibroid phthisis is based chiefly on the absence of extensive fibrosis and contraction of the

lung which are characteristic for the cirrhotic-indurative forms of chronic pulmonary tuberculosis. The distinction between cystic bronchiectasis and tuberculous parenchymal cavities rests on the typical sponge-like structure, the multilocular thin-walled appearance of the annular formations and the absence of any changes indicative of an infiltrative process in the former. The persistent absence of tubercle bacilli from the sputum is, of course, a decisive point against tuberculosis, especially in view of the roentgen findings indicative of cavitations. It should be mentioned, however, that occasionally tuberculosis may be a complicating feature of cystic bronchiectasis.

(3) *Emphysematous Bullae.* From a roentgenographic standpoint only multiple small or medium sized bullae may have to be considered in a differential diagnosis of cystic bronchiectasis. Large or so-called "giant" emphysematous bullae resemble more closely the large "balloon" type of pulmonary cyst. In contrast to cystic bronchiectasis, emphysematous bullae rarely show well circumscribed complete ring shadows. The presence of multiple fluid levels, often found in cystic bronchiectasis, speaks against emphysematous bullae. It should be kept in mind, however, that a more or less marked degree of diffuse emphysema, with or without demonstrable bulla formation, is not infrequently associated with cystic bronchiectasis, particularly in older individuals.

(4) *Other forms of cystic disease of the lung*, especially large solitary or multiple cysts, are found mostly in infants and young children, whereas cystic bronchiectasis may be observed in any age. The former usually cause quite marked symptoms of respiratory distress with signs and symptoms of mediastinal pressure. The physical and roentgen findings are very similar to those observed in pneumothorax. Signs of intrathoracic pressure, such as displacement of the heart and mediastinal organs to the opposite side, are often present. It is evident that there is very little similarity between changes of this type and the characteristic findings observed in cystic bronchiectasis.

COURSE, PROGNOSIS AND THERAPY

Our observations in this group of cases would seem to indicate that the clinical course is, on the whole, of a non-progressive character and that the prognosis is rather favorable, provided that no complications develop. In younger persons the presence of cystic bronchiectasis is apparently compatible with general good health and with complete or nearly complete absence of subjective symptoms. However, with advancing age some of these individuals become chronic sufferers because of recurrent infections which may eventually set up a chronic suppurative process. An originally harmless anatomic abnormality is thus transformed into a serious and at times disabling illness.

Other possible complications of clinical significance are pulmonary emphysema and cardiac insufficiency, especially right heart failure. The latter may be the result of the chronically impaired pulmonary circulation caused by extensive pulmonary changes such as are seen in some cases of cystic bronchiectasis. The frequently associated emphysema is an additional functional burden on the right heart. Of the cases included in this report, 2 patients aged fifty-seven and seventy years respectively died from cardiac insufficiency. In these cases, however, the cardiovascular complication was apparently independent of the pulmonary lesion.

As to the therapy, it is evident that cases of uncomplicated cystic bronchiectasis presenting no symptoms require no particular treatment. Certain preventive measures, however, may be of some advantage. These individuals should be aware of their condition and should be advised to take proper care of apparently harmless respiratory infections. Otherwise, the treatment is purely symptomatic. Cases in which the original lesion is complicated by severe chronic infection should be treated along the same

lines as any other chronic suppurative bronchiectasis. Needless to say, cases of cystic bronchiectasis present no indication for surgical measures, such as lobectomy, unless complicated by a progressive suppurative process.

A final point to be emphasized is the fact that in the past these individuals have apparently often been regarded as suffering from pulmonary tuberculosis. Many of them have gone from one tuberculosis institution to another where they remained for varying lengths of time, frequently over a period of years. The unnecessary hardship as well as the hazard of exposure to tuberculous infection thus imposed on these individuals is quite obvious. The needless financial burden to the community may also be a point worthy of consideration in this connection.

REPORT OF CASES

A. Uncomplicated cystic bronchiectasis. Clinical symptoms absent or very mild (Cases I to IX).

Case I. J. M., a white boy, was first seen in one of the Tuberculosis Clinics of the Department of Health at the age of fourteen. His only complaints were frequent colds and occasional mild non-productive cough. Examination of the chest showed dullness, many coarse moist râles over the right upper lobe and few râles at the right base. There were no other significant findings. During the subsequent follow-up period of three years, part of which he had spent in a tuberculosis hospital, he was practically symptom-free. The sputum was always negative for tubercle bacilli. Roentgen examination disclosed a honeycomb-like structure of the right upper lobe due to multiple small thin-walled cavities without surrounding infiltration or fibrosis. There was no retraction of the lung field or distortion of the adjacent structures. On bronchographic examination the annular formations could be identified as a system of saccular dilatations of the bronchi which filled readily with the contrast material. Some of the larger bronchial branches showed fusiform widening (Fig. 1).

Case II. J. T., a white boy, aged eleven, was admitted to Sea View Hospital with a history of mild non-productive cough since the age of one. There were no other significant data in the history. Examination of the chest disclosed numerous medium and coarse moist râles over the right lung especially at the base. The sputum was negative for tubercle bacilli on numer-

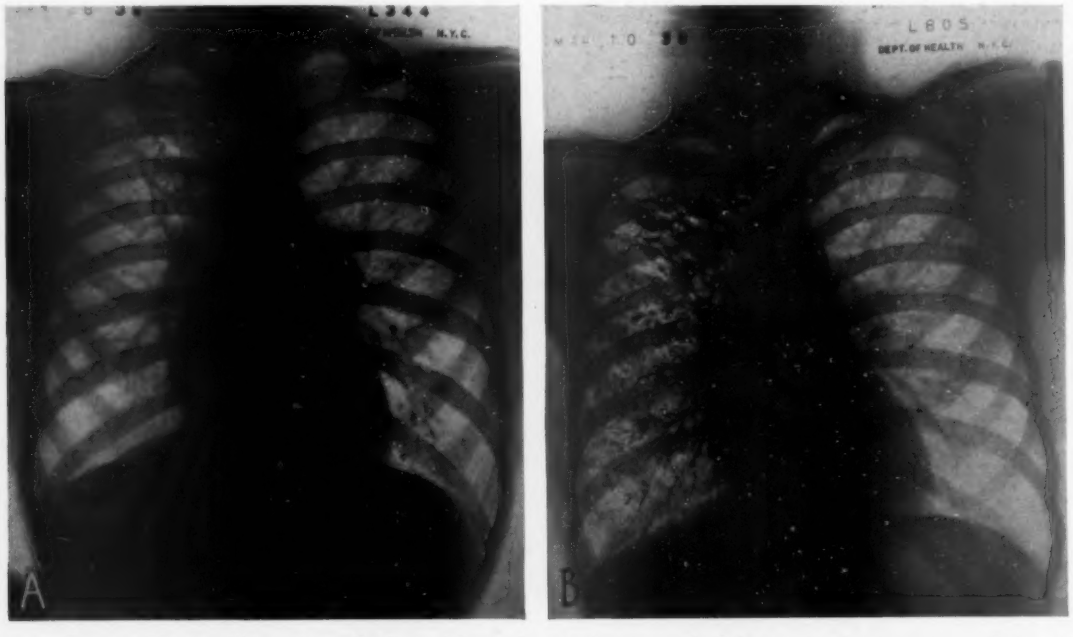

Fig. 1. Case I. Uncomplicated cystic bronchiectasis. *A* shows typical honeycomb structure of the right upper lobe. Bronchogram (*B*) demonstrates a system of saccular bronchial dilatations and fusiform widening of the larger bronchial branches in the right upper lobe.

ous occasions. He left the hospital after sixteen months having no subjective symptoms. Roentgen examination showed a system of multiple thin-walled cavities in the lower half of the right lung with no apparent impairment of aeration or retraction of the lung field. He was seen again at the age of twenty, after a lapse of nearly eight years, when he stated that he has had no subjective symptoms and that he has been able to pursue a perfectly normal mode of life. A recent roentgenogram showed that some of the cavities had increased in size and that they contained small fluid levels. Bronchography disclosed a number of saccular and cylindrical dilatations of the bronchi in the right lower lobe and few in the middle lobe (Fig. 2).

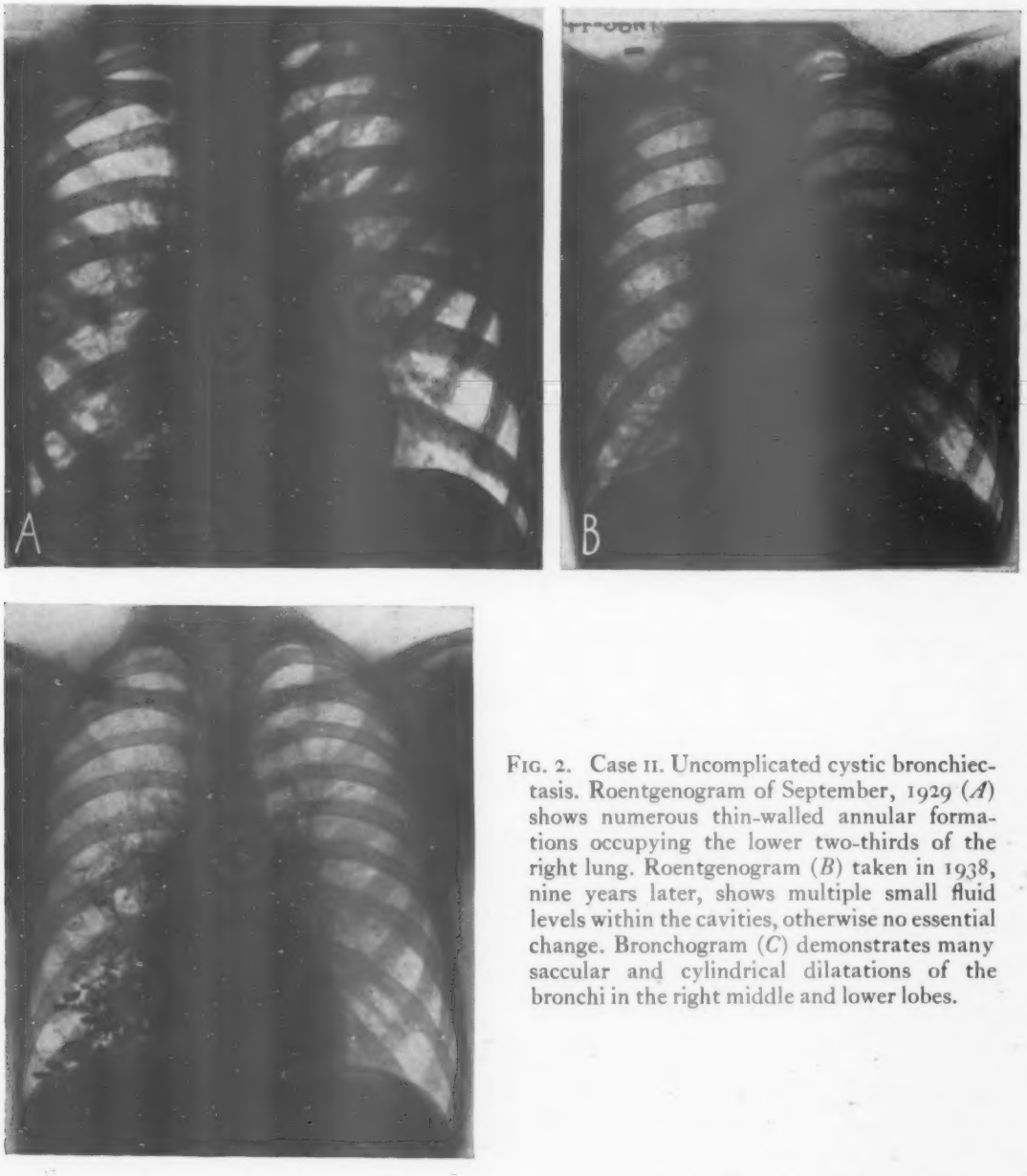

Fig. 2. Case II. Uncomplicated cystic bronchiectasis. Roentgenogram of September, 1929 (*A*) shows numerous thin-walled annular formations occupying the lower two-thirds of the right lung. Roentgenogram (*B*) taken in 1938, nine years later, shows multiple small fluid levels within the cavities, otherwise no essential change. Bronchogram (*C*) demonstrates many saccular and cylindrical dilatations of the bronchi in the right middle and lower lobes.

Case III. V. F., a white boy, aged thirteen, was a contact to a brother who had pulmonary tuberculosis and was therefore examined in one of the Tuberculosis Clinics of the Department of Health. He gave a history of frequent colds and cough with slight mucoid expectoration since early childhood. Examination of the chest

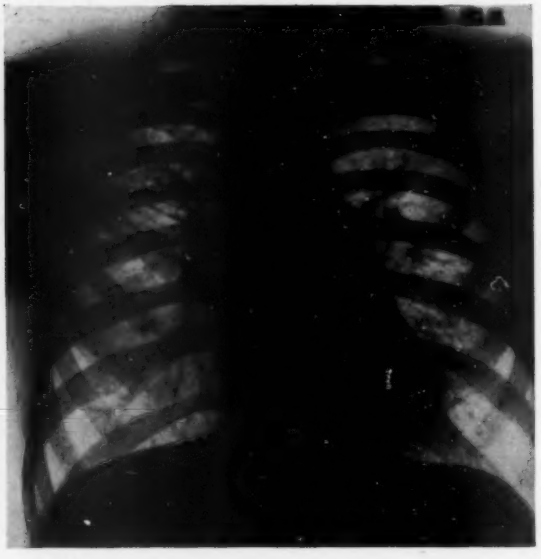

Fig. 3. Case III. Uncomplicated cystic bronchiectasis. Characteristic sponge-like structure of the right upper lobe due to a cluster of multiple thin-walled cavities.

showed dullness, bronchovesicular breathing and many medium moist râles over the right upper lobe; few râles were also present at the right base. The Mantoux test was negative and the sputum showed no tubercle bacilli. Roentgen examination disclosed a characteristic spongy appearance of the right upper lobe which consisted of a network of thin annular formations without infiltration or fibrosis (Fig. 3). Bronchographic examination showed some saccular collections of contrast material in the right upper lobe and a few also at the base of the right lung.

Case IV. V. G., a white female, aged twenty-seven, was examined routinely in the course of a mass case-finding survey for pulmonary tuberculosis. She has had frequent chest colds and a chronic mild cough without expectoration. Her past history was otherwise irrelevant. Examination of the chest disclosed dullness and moist râles over the right upper lobe, otherwise no significant findings. The sputum was negative for tubercle bacilli. Roentgen examination revealed a honeycomb structure of the right upper lobe consisting of a system of thin-walled cavities some of which showed fluid levels. There was no evidence of infiltration, fibrosis or retraction of the lung field.

Case V. L. F., a white woman, aged thirty-six, gave a history of "bronchitis" since early childhood. She was examined in the outpatient department of a general hospital where it was thought that she had pulmonary tuberculosis and she was therefore admitted to Sea View Hospital. Her past history was irrelevant. On admission she appeared to be in good general health and was entirely symptom-free. Examination of the chest revealed many moist râles and rhonchi over the entire right lung. Otherwise, there were no noteworthy findings. The laboratory data did not show anything of significance. Sputum could not be obtained for examination. She felt perfectly well and left the institution after a short stay. Roentgen examination showed numerous thin-walled annular formations throughout the right lung ranging from cherry to walnut size. In the basal portion small fluid levels were present within the cavities. The lung field appeared well aerated and there was no evidence of shrinkage of the pulmonic field or retraction of the adjacent structures.

Case VI. M. S., a white female, aged thirty, visited a maternity clinic because of pregnancy. A routine physical examination of the chest revealed signs which suggested tuberculosis and she was advised to enter the Tuberculosis Division of the Metropolitan Hospital. On admission she stated that she had had a slight non-productive cough for about one year. Her past history was irrelevant except for pneumonia in childhood. She appeared to be in good general health. Examination of the chest showed dullness, bronchovesicular breathing and numerous moist râles over the right upper lobe; a moderate number of râles were also present at both bases. There were no other significant findings. The sputum was negative for tubercle bacilli. A blood Wassermann test was positive. Roentgen examination of the chest revealed a typical spongy appearance of the right upper lobe due to numerous closely packed thin-walled cavities with marked overlapping of their borders. Small collections of fluid were seen in some of the cavities. There was no shrinkage of the lung field or retraction of the mediastinal shadow. Bronchographic studies disclosed a system of saccular bronchial dilatations in the right upper lobe. Saccular and cylindrical bronchiectasis was also present at the base of the left lung and few at the right base. She was followed for a period of six and one-half years during which time she remained

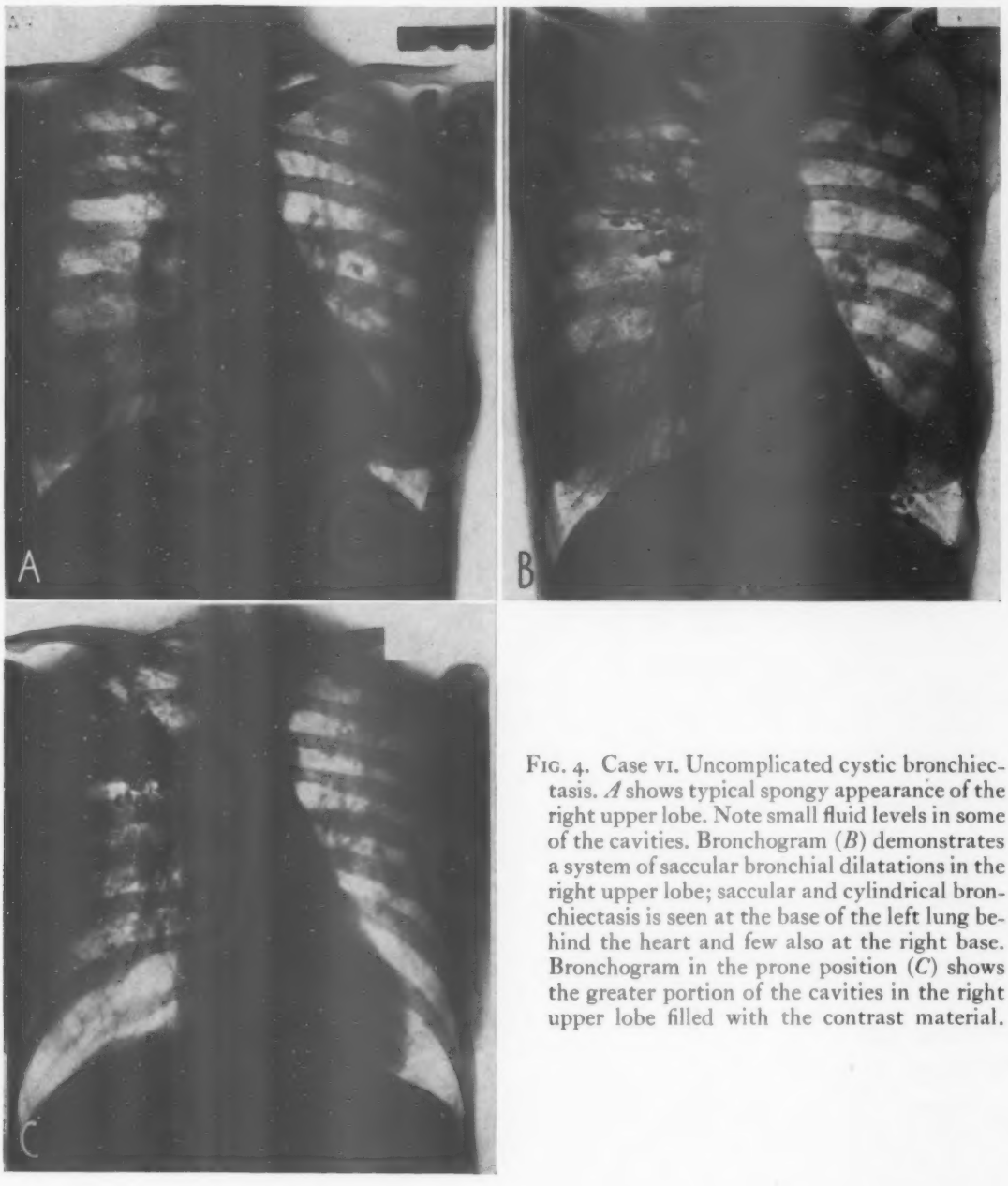

Fig. 4. Case vi. Uncomplicated cystic bronchiectasis. *A* shows typical spongy appearance of the right upper lobe. Note small fluid levels in some of the cavities. Bronchogram (*B*) demonstrates a system of saccular bronchial dilatations in the right upper lobe; saccular and cylindrical bronchiectasis is seen at the base of the left lung behind the heart and few also at the right base. Bronchogram in the prone position (*C*) shows the greater portion of the cavities in the right upper lobe filled with the contrast material.

practically symptom-free. There was no change in the roentgen findings throughout the entire period (Fig. 4).

Case vii. E. R., a white male, aged twenty-six, was first seen in one of the Tuberculosis Clinics of the Department of Health in 1931. In 1929 he had been told that he had pulmonary tuberculosis, evidently on the basis of a physical examination. At no time did he have symptoms referable to the respiratory tract. His past history was irrelevant. Examination of the chest showed dullness, bronchovesicular breathing, increased vocal fremitus and moist râles over the left upper lobe. Otherwise there were no significant findings. He was observed for a period of seven years until August, 1938, during which time he failed to show any subjective symptoms. The objective findings remained essentially unchanged. The sputum was persistently negative for tubercle bacilli. Roentgen examination showed a honeycomb

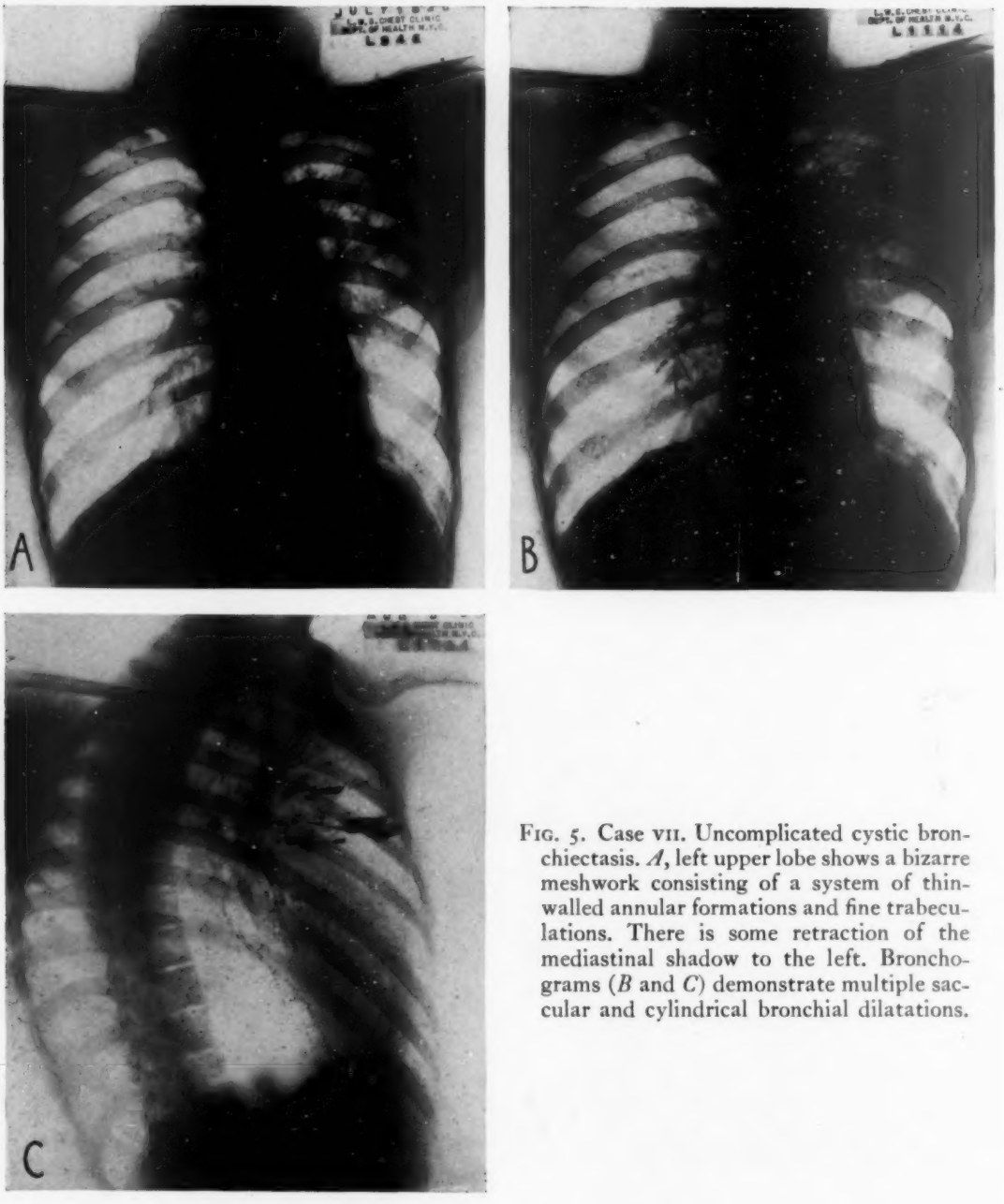

Fig. 5. Case vii. Uncomplicated cystic bronchiectasis. *A*, left upper lobe shows a bizarre meshwork consisting of a system of thin-walled annular formations and fine trabeculations. There is some retraction of the mediastinal shadow to the left. Bronchograms (*B* and *C*) demonstrate multiple saccular and cylindrical bronchial dilatations.

appearance of the left upper lobe from the apex to the third anterior rib with numerous thin-walled annular shadows and fine linear trabeculations. The superior mediastinum showed some retraction to the left and the intercostal spaces were somewhat narrowed in the involved area. The remaining lung fields presented an emphysematous appearance. Bronchographic studies revealed multiple saccular collections of contrast material as well as cylindrical bronchiectasis leading directly into the saccular dilatations (Fig. 5).

Case viii. T. M., a white male, aged thirty-three, was admitted to Metropolitan Hospital complaining of abdominal cramps and diarrhea of several weeks' duration. He had no past history or symptoms indicative of a respiratory affection. Examination of the chest revealed slight dullness, few rhonchi and fine moist

râles over the right upper lobe. There were no other significant findings. The sputum was negative for tubercle bacilli. A blood Wassermann test was positive. Roentgen examination of the chest showed a number of thin-walled cavities occupying the right upper lobe, some containing small collections of fluid. There was no evidence of fibrosis or shrinkage, the mediastinal shadow was in normal position. Bronchographic study revealed multiple saccular and cylindrical bronchial dilatations in the right upper lobe and few saccular dilatations at the base of the right lung.

Case IX (courtesy of Dr. D. Ulmar). J. H., a white male, aged twenty-four, had a past history of frequent colds. He complained of pain in the right lower chest and slight nonproductive cough of several weeks' duration. On physical examination bronchovesicular breathing and medium moist râles were found over the right base anteriorly. There were no other abnormal findings. Roentgenograms of the chest showed multiple thin-walled cavities occupying the right middle lobe some of which contained small collections of fluid. There was no retraction of the involved lobe or of the adjacent structures. During the subsequent follow-up period of eighteen months he had no subjective symptoms except a slight cough. The physical signs and the roentgen findings remained unchanged. The sputum was persistently negative for tubercle bacilli.

B. Cystic bronchiectasis complicated by chronic infection. Clinical symptoms moderate to severe. Roentgenological signs of associated chronic inflammatory changes (*Cases X to XII*).

Case X. J. C., a white male, aged forty-seven, was observed at the Metropolitan Hospital for a period of nine years. He gave a history of cough associated with slight expectoration of at least twenty years' duration. These symptoms were subject to seasonal exacerbations during the winter months. During the last few years the symptoms became more marked, the cough was more persistent, he expectorated moderate amounts of purulent sputum and developed progressive dyspnea on exertion. There was, however, no history of

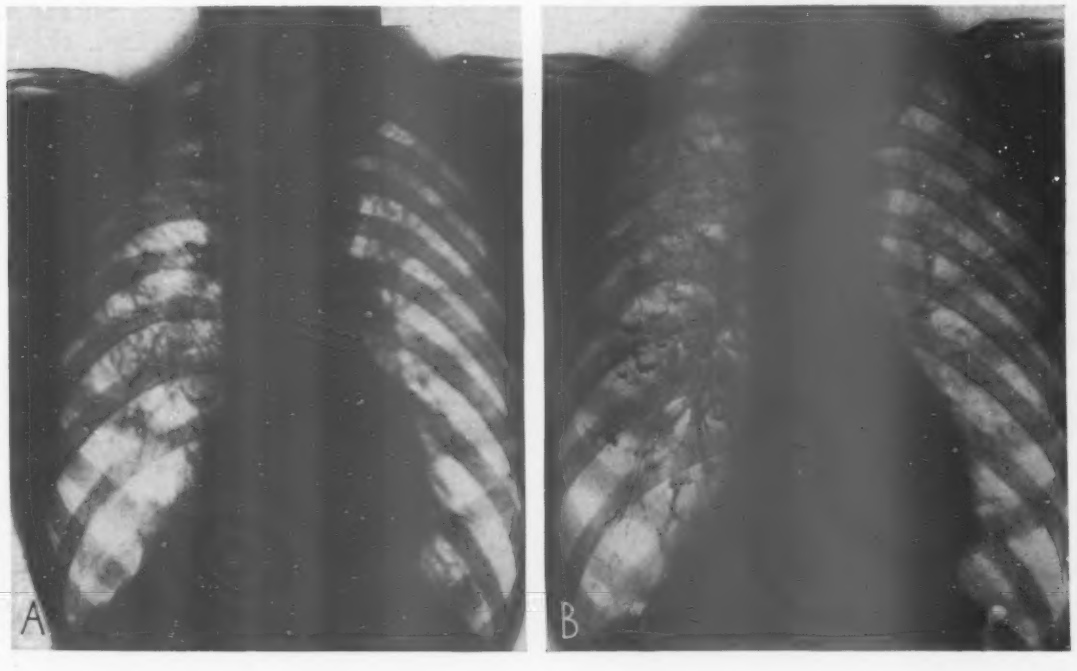

Fig. 6. Case X. Cystic bronchiectasis complicated by chronic infection. *A* shows multiple thin-walled cavities occupying the upper two-thirds of the right lung. Note fibrosis and signs of shrinkage in the superior portion of the upper lobe. Diffuse emphysema of the remainder of the lung fields. Bronchogram (*B*) demonstrates a system of saccular bronchial dilatations in the right upper and middle lobes and fusiform widening of the descending branches.

Fig. 7. Case xi. Cystic bronchiectasis complicated by chronic infection. Sponge-like structure of nearly the entire right lung and of the left basal portion. Changes indicative of a chronic inflammatory process are present at the bases, especially on the left side.

copious or bloody expectoration. It may be of interest to mention that he had spent the greater part of the twenty years in various tuberculosis institutions. On physical examination there was some retraction of the right hemithorax, dullness, bronchovesicular to bronchial breathing over the right upper lobe, many medium and coarse moist râles over the greater portion of the right lung and signs of emphysema at both bases. There was slight clubbing of the fingers. The physical findings remained essentially the same during the entire period of observation. His general condition appeared to be fairly stationary. The sputum never contained tubercle bacilli. Other laboratory findings were of no significance. Roentgen examination showed that the upper and middle lobes of the right lung were occupied by multilocular thin-walled cavities of pea to walnut size. Small fluid levels were seen in some of the cavities. In the upper portion of the right upper lobe there was a moderate degree of fibrosis with narrowing of the intercostal spaces and retraction of the trachea to the right. The remaining lung fields showed diffuse emphysema. These findings remained essentially the same throughout the follow-up period. Bronchography demonstrated that the described cavities represented saccular bronchial dilatations communicating freely with the major bronchial branches. There was also cylindrical and fusiform dilatation of the bronchi in the right upper and middle lobes (Fig. 6).

Case xi. E. G., a white male, aged forty-eight, presented a history very similar to the preceding case, that is, a mild chronic cough with scanty expectoration and seasonal exacerbations for about twenty years. These symptoms increased in severity during the more recent years simultaneous with development of progressive dyspnea. For many years he had been a patient in various tuberculosis hospitals and was observed at Metropolitan Hospital for a period of nine years. Examination revealed moderate dyspnea and cyanosis and slight clubbing of the fingers. There were signs of diffuse emphysema and numerous medium and coarse râles throughout both lungs as well as many rhonchi at the bases. The laboratory data showed no significant findings. The sputum was always negative for tubercle bacilli. Roentgen examination showed that the greater portion of the right lung was occupied by a bizarre network composed of numerous closely packed thin-walled cavities producing a characteristic sponge-like appearance. The left lung showed emphysema in the upper two-thirds and a honeycomb structure in the basal portion, similar to the corresponding area on the right side, with changes suggestive of a chronic inflammatory process. The linear densities in the left upper portion suggested calcified pleural plaques. Bronchographic examination was not performed (Fig. 7).

Case xii. P. O., a white male, aged fifty-four, has been under observation at the Metropolitan and Sea View Hospitals for a period of eleven years. His history and symptoms closely resembled those of the preceding two cases. He had a chronic cough and scanty expectoration of many years' duration with seasonal exacerbations, increasing severity of symptoms in recent years and progressive dyspnea. He, too, had been a patient in various tuberculosis institutions for many years. Examination of the chest

revealed dullness over the right upper lobe, bronchovesicular breathing, many moist râles and rhonchi over the right upper and middle lobes. The sputum was persistently negative for tubercle bacilli. There were no other laboratory findings of significance. Roentgen examination showed numerous cavities of various sizes in the right upper and middle lobes, the largest being about the size of a walnut. The mid-zone presented a characteristic honeycomb structure while the upper portion of the lung showed definite fibrosis and infiltration suggestive of an associated chronic inflammatory process. The remainder of the right lung as well as the left lung presented an emphysematous appearance.

C. Cystic bronchiectasis complicated by pulmonary tuberculosis (Case XIII).

Case xiii. J. P., a white female, aged thirty-four, applied for admission to the obstetric ward of Metropolitan Hospital because of pregnancy. She had no history or symptoms referable to the respiratory tract except occasional chest colds. She was transferred to the Tuberculosis Division because a routine examination disclosed abnormal physical signs in the chest which suggested pulmonary tuberculosis. Her general condition was good, constitutional signs and symptoms were absent. Examination of the chest showed numerous medium and coarse moist râles throughout both lungs from apex to base. There were no other significant findings on physical examination. The sputum, which at first contained tubercle bacilli on several occasions, became persistently negative two months after admission. Bronchoscopy did not disclose any abnormal findings. Roentgen examination revealed a sponge-like structure of both lung fields due to numerous thin-walled cavities with occasional fluid levels. In spite of the extensive changes the pulmonic fields appeared to be well aerated, except in the left apex which showed a diffuse density with an area suggestive of a small cavity. The latter findings were interpreted as due to a complicating tuberculous lesion. Bronchography disclosed numerous saccular and cylindrical dilatations of the bronchi in the left lung. A subsequent attempt to visualize the bronchial tree on the right side was unsuccessful. The patient was observed in the institution for a period of six months. She remained entirely symptom-free and was discharged in good general condition.

D. Cystic bronchiectasis complicated by cardiovascular disease, apparently not related to the pulmonary lesion (Cases XIV and XV).

Case xiv. G. A., a white male, aged fifty-seven, was admitted to Sea View Hospital complaining of progressive dyspnea on exertion and distressing cough with scanty expectoration of several months' duration. He stated that seventeen years before he had consulted a physician because of cough and expectoration and that bronchiectasis was diagnosed at that time. He was then apparently well until a few months before admission to the hospital. Physical examination revealed bronchovesicular breathing and many coarse moist râles over the right upper lobe and rhonchi at both bases. The heart was enlarged to the left. The blood pressure was 170/80. Roentgen examination of the chest showed a honeycomb structure of the right upper lobe due to a number of thin-walled cavities some of which contained small fluid levels. The increased hilar shadows and the prominent peripheral markings suggested vascular stasis. On the roentgenogram the latter findings obscured to some extent the other changes in the right upper lobe (Fig. 8*A*). During the subsequent course the patient developed symptoms and signs of progressive circulatory congestion and eventually died as a result of cardiac insufficiency. The sputum was always negative for tubercle bacilli. Other laboratory data were essentially irrelevant.

The autopsy findings were as follows: Marked hypertrophy and dilatation of the heart, especially of the left ventricle; fibrosis of the myocardium; mitral insufficiency; marked arteriosclerosis of the aorta and sclerosis of the coronary arteries; congestion of all organs. Both lungs were markedly anthracotic, with the exception of the right upper lobe. The light yellowish-white color of the latter presented a striking contrast to the dark discoloration of the remainder of the lungs. On section the right upper lobe showed a sponge-like appearance and consisted of numerous thin-walled cyst-like cavities ranging between a few millimeters and 2 cm. in diameter. There were also many cylindrical and fusiform bronchial dilatations some of which terminated in the cystic dilatations. There was slight increase in fibrous tissue between the bronchial dilatations. Whatever lung parenchyma was present in this lobe appeared to be markedly emphysematous. The pleura over the right upper lobe was thickened.

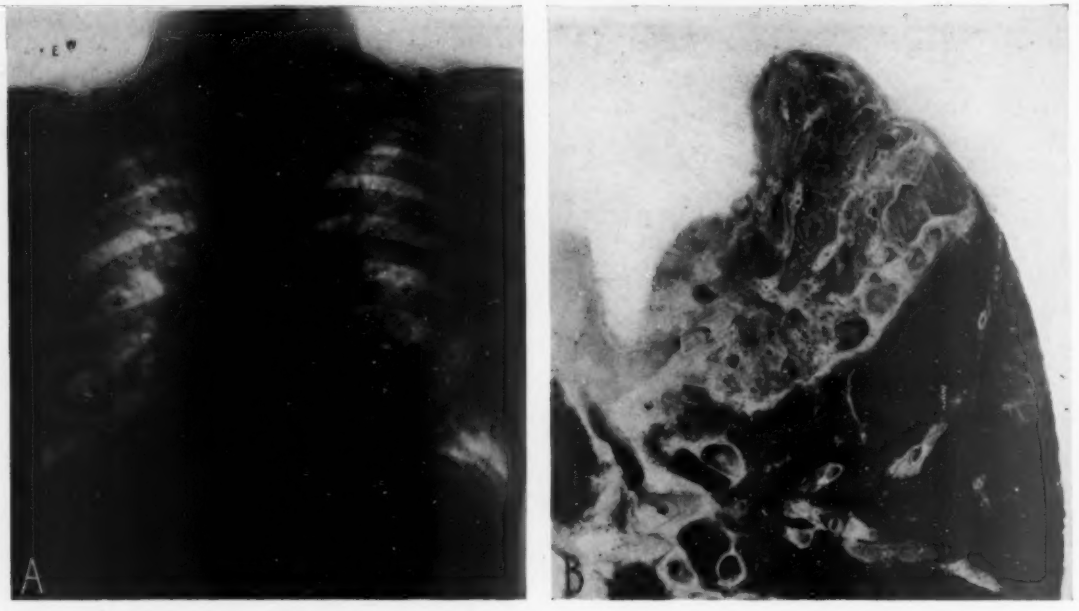

FIG. 8. Case XIV. Cystic bronchiectasis complicated by cardiovascular disease. Roentgenogram (*A*) shows a honeycomb structure of the right upper lobe due to multiple thin-walled cavities. The vascular markings are prominent throughout as a result of circulatory congestion. Postmortem specimen of the lung (*B*) showing the right upper lobe and the adjoining portion of the lower lobe. The upper lobe shows a spongy appearance and consists of numerous cyst-like saccular and fusiform bronchial dilatations. Note the light color of the upper lobe as it contrasts with the dark anthracotic pigmentation of the adjacent portion of the lower lobe.

The other pulmonary lobes showed marked anthracosis, vascular congestion and terminal bronchopneumonia (Fig. 8*B*). Microscopic examination showed that the walls of the cyst-like cavities consisted of muscle, cartilage, mucous glands and a small amount of fibrous tissue. Their inner surface was lined by cuboidal epithelium. In places the mucous membrane was replaced by vascular granulation tissue. The septa between the cavities consisted of fibrous tissue with some fibroblasts and lymphocytes. There was only a very slight amount of coal pigment in the apex of the right upper lobe, otherwise this lobe was free of anthracotic deposits.

CASE XV. L. K., a white male, aged seventy, was admitted to Sea View Hospital with a history of chronic cough and expectoration of scant mucopurulent sputum since early childhood. During the last two years these symptoms had increased in severity. He developed progressive dyspnea on exertion and pain in the chest radiating to the shoulder. One week before admission he had a slight hemoptysis. On examination he appeared to be markedly dyspneic and orthopneic and showed pronounced cyanosis. There was clubbing of the fingers. The chest was of an emphysematous type. There was no impairment of resonance on percussion. There were numerous coarse moist râles over the right lung and many rhonchi over both lungs. The heart appeared to be enlarged to the left. The peripheral vessels were sclerotic. The blood pressure was 200/90. The liver was enlarged and tender on palpation. During the subsequent course he showed signs and symptoms of progressive circulatory failure. He died eight months after admission to the hospital as a result of cardiac insufficiency. The sputum was always negative for tubercle bacilli. There were no other significant laboratory findings. Roentgen examination of the chest showed a characteristic sponge-like structure of almost the entire right lung field, due to numerous thin-walled cavities ranging between cherry and walnut size, some of which contained small fluid levels. There was no evidence of shrinkage of the lung; the mediastinum, the heart and the diaphragm were in normal position. The left lung showed marked emphysema. The heart was generally enlarged, especially to the left. Permission for autopsy was not obtained.

E. Cases with massive involvement of one lung (Cases XVI and XVII).

Case XVI. L. C., a white female, aged thirty-six, was admitted to the Tuberculosis Division of Metropolitan Hospital in July, 1933. Her chief symptoms were chronic cough with slight expectoration since the age of five. She had had a hemoptysis on one occasion some years before admission. Her past history was otherwise irrelevant. She had been a patient in several tuberculosis institutions for several years. For a period of five years before admission to the hospital she felt well and had no symptoms except a moderate cough. She became pregnant and applied for admission to the hospital because she had previously been told that she had pulmonary tuberculosis. On admission she appeared to be in good general condition. Examination of the chest disclosed diminished respiratory expansion of the left side with marked dullness and many coarse moist râles over the entire left lung. The heart appeared to be displaced to the left. Otherwise there were no noteworthy findings. The sputum was negative for tubercle bacilli on many occasions. The other laboratory examinations showed nothing of significance. Roentgen examination showed a sponge-like appearance of the entire left lung field due to multilocular rarefactions of various sizes, the largest being located in the apical portion. There was no normal lung structure seen anywhere on the left side. The heart and the mediastinal shadow were entirely within the left hemithorax and the intercostal spaces were moderately narrowed on this side. The right lung showed marked emphysema. Bronchographic examination showed numerous saccular as well as cylindrical and varicose bronchiectases on the left side. It may be of interest to point out that the contrast material did not enter the large cavity situated in the apex, possibly because of a block in the bronchial lumen. The patient was discharged from the hospital in June, 1934, in good general condition. She was seen again four years later when she stated that she had been symptom-free during this period except for a slight cough. The roentgen findings were essentially the same as above except that within the large apical cavity there could be noted a dense sharply circumscribed opacity of ovoid shape. While no definite explanation can be offered for the latter finding, it is suggested that it may possibly be due to a blood clot within the cavity (Fig. 9).

Case XVII. J. L., a colored male, aged fifty, was admitted to Rikers Island Hospital complaining of frequent "chest colds" and cough with very little expectoration of three years'

Fig. 9. Case XVI. Cystic bronchiectasis with massive involvement of the left lung. Roentgenogram of July, 1933 (*A*) shows sponge-like structure of the entire lung field due to multilocular rarefactions. Note large cystic formation occupying the apical and subapical area. Marked retraction of the mediastinum and heart to the left. Bronchogram (*B*) demonstrates multiple saccular and some fusiform bronchial dilatations in the lower two-thirds. *C* (July, 1938) shows the appearance of the left lung five years after the original roentgenogram (Potter-Bucky technique). Note density of ovoid shape within the large upper cyst.

duration. About ten years before admission he had had pneumonia on the right side, otherwise his past history was irrelevant. Examination showed flattening of the left side of the thorax and dullness on percussion. Over the left lung the breath sounds were bronchial to amphoric and there were numerous sibilant and sonorous râles. The heart appeared to be displaced to the left, the right border being at the left margin of the sternum. There was a systolic murmur over the apex and the second pulmonic sound was accentuated. The rest of the physical examination was essentially negative. He remained under observation for a period of six weeks and at the time of discharge was entirely symptom-free. The sputum was negative for tubercle bacilli. There were no other laboratory findings of significance. Roentgen examination disclosed changes of the left lung which were almost a replica of the previous case, that is, a spongy structure with a network consisting of coarse meshes, suggesting a system of closely packed cavities. Small fluid levels were seen in some of the cavities. As in the other case, no normal parenchyma could be seen on this side. There was marked retraction of the mediastinum and heart to the left with narrowing of the intercostal spaces and elevation of the left side of the diaphragm. The right lung was markedly emphysematous. Bronchographic examination was refused by the patient.

SUMMARY AND CONCLUSIONS

(1) A clinical and roentgenological study of a group of seventeen cases of cystic bronchiectasis is presented.

(2) The findings observed in these cases indicate that this lesion represents a distinct entity which should be separated both from the common forms of saccular bronchiectasis and from other types of cystic disease of the lung.

(3) The clinical symptomatology and findings are described. The frequent disproportion between the paucity of subjective symptoms and the extensive pulmonary changes is stressed. At the same time it is also brought out that in a certain proportion of the cases secondary infection may result in a chronic suppurative process.

(4) The roentgen findings of the honeycomb- or sponge-lung characteristic of cystic bronchiectasis are described.

(5) The pathology and pathogenesis are discussed with particular reference to the question of the congenital origin of the lesion.

(6) The clinical and roentgenological differentiation of cystic bronchiectasis from saccular bronchiectasis of inflammatory origin, tuberculosis, emphysematous bullae and other forms of cystic disease of the lung is discussed.

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VENTRICULOGRAPHIC LOCALIZATION OF INTRACRANIAL TUMORS*

III. TUMORS OF THE CEREBELLUM AND FOURTH VENTRICLE

By VINCENT C. JOHNSON, M.D., *and* CARL F. LIST, M.D.
ANN ARBOR, MICHIGAN

VENTRICULOGRAPHIC signs characteristic of tumors arising in the aqueduct, pons and cerebellopontine angle have been discussed in a previous communication.[1] The purpose of this paper is to describe the ventriculographic signs produced by tumors involving the cerebellum and fourth ventricle. Since the surgical and roentgenographic technique of ventriculography has already been described in the first paper[2] of this series, it is unnecessary to again discuss our methods.

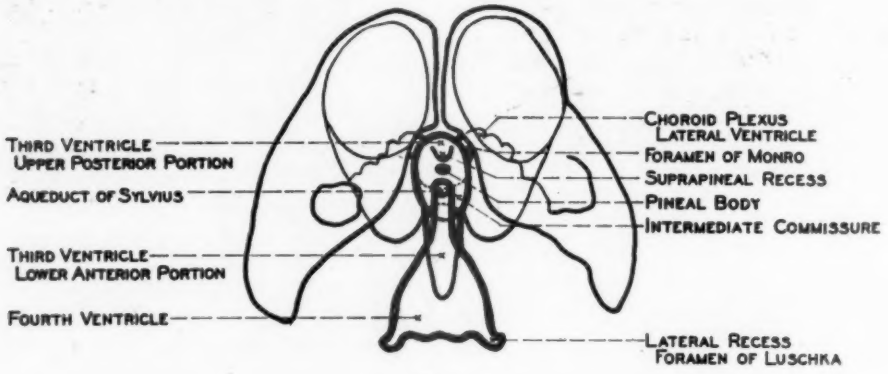

FIG. 1

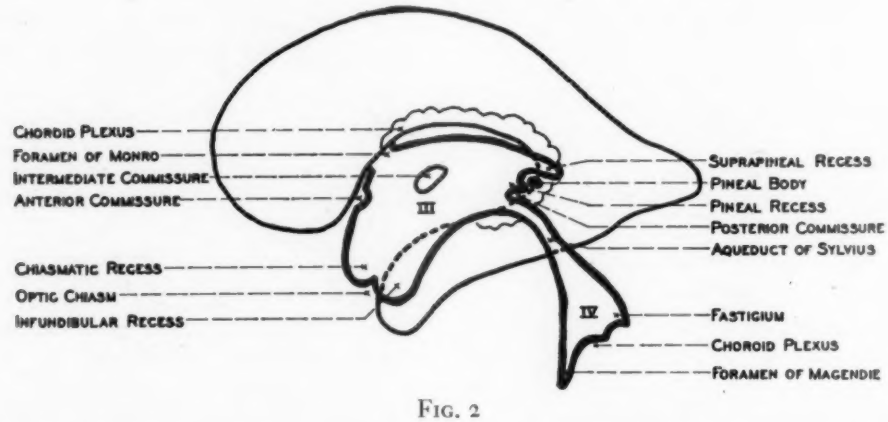

FIG. 2

It has been previously stated that changes in ventriculographic appearance pathognomonic of posterior fossa tumors concern the aqueduct and fourth ventricle. Compression of these structures results in varying degrees of internal hydrocephalus always associated with tumors in this location.

The position and shape of the aqueduct and fourth ventricle, when moderate dilatation of the ventricular system exists, have already been described. Figures 1 and 2 are again reproduced to demonstrate these relationships.

Tumors involving the posterior fossa are either extra- or intraventricular in type.

* From the Department of Roentgenology and the Department of Surgery, Service of Dr. Max M. Peet, University of Michigan, Ann Arbor, Michigan.

The extraventricular group includes midline vermis tumors and lateralized hemispheral tumors. Although subtentorial meningiomas of the transverse sinus arise outside of the cerebellum, they will be included in the discussion of lateralized

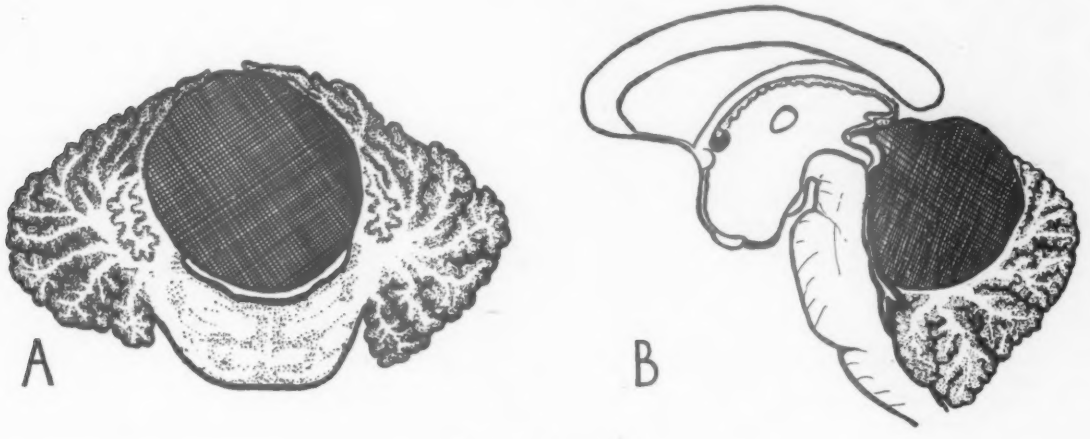

Fig. 3. *A* and *B*.

intracerebellar tumors, since the ventriculographic deformities which they may produce are similar. The intraventricular group consists entirely of those tumors which are situated completely within the fourth ventricle.

I. Extraventricular Tumors.

a. Midline Cerebellar Tumors. Deformity and displacement of the aqueduct and fourth ventricle produced by these lesions are demonstrated by schematic drawings of the verticofrontal section (Fig. 3*A*), and the mid sagittal section (Fig. 3*B*) of a cerebellum containing a tumor of the upper vermis. The tumor represented by the densely cross-hatched area is seen encroaching upon the aqueduct and fourth ventricle from its dorsal midline position.

If the tumor occupies the dorsal vermis and extends high into the incisura tentorii, the aqueduct may be compressed at its beginning, thus simulating the ventricular deformity produced by a true neoplasm of the aqueduct (Fig. 4, Case 1). Tumors of the dorsal vermis, usually astrocytomas, may cause a slight anterior and superior displacement of the inferior outline of the third ventricle due to shift of the brain stem and occasionally by pressure or invasion directed upward through the incisura of the tentorium. Thus, in Figure 4, it will be noted that the inferior margin of the third ventricle is displaced slightly upward, since this margin is not superimposed by the temporal horn, as should be the case with this degree of internal hydrocephalus.

If the tumor is situated in the lower vermis (near the foramen magnum) (Fig. 5,

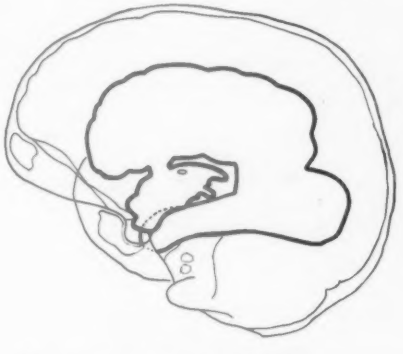

Fig. 4. Case 1 (352200).

Case 11), not only the aqueduct but also a part of the fourth ventricle may be visualized. At operation, Case 11 was found to have a hemangioblastoma, with a large cyst in the tonsillar region. The triangular roof portion of the fourth ventricle is usually flattened by the tumor mass in this region, and only a thin streak of oxygen may

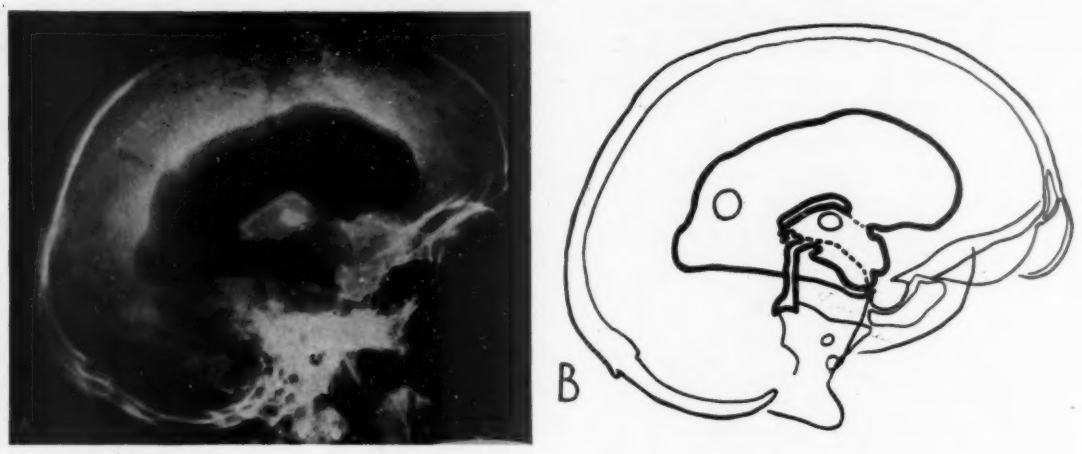

Fig. 5.* *A* and *B*. Case 11 (321054).

be seen outlining the floor of the fourth ventricle. Such tumors clearly show the anterior displacement of the pons in the lateral views. The aqueduct forms an angle of 70–120 degrees with the anterior outline (floor) of the fourth ventricle, instead of the normal angle of more than 130 degrees. This type of deformity is further demonstrated by a schematic drawing of a mid sagittal section illustrating a low vermis tumor (Fig. 6).

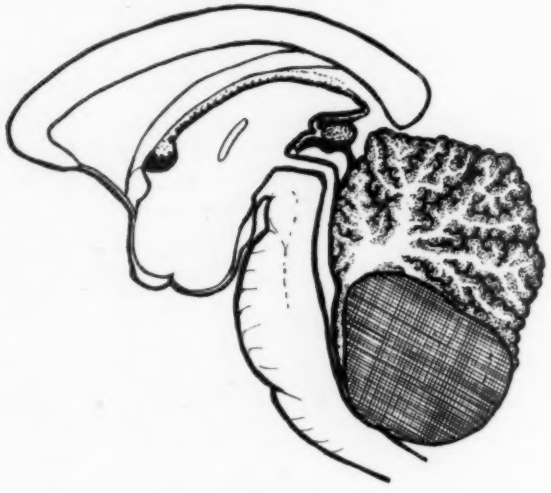

Fig. 6

The sagittal ventriculogram, with the occiput superior, is essential to demonstrate the midline position of these tumors; hence, in this projection the aqueduct or fourth ventricle is always visualized in the midline. Displacement of these structures to either side is characteristic of lateralized cerebellar lesions.

* The outlines of the third ventricle, aqueduct and fourth ventricle have been accentuated by retouching in all reproduced roentgenograms.

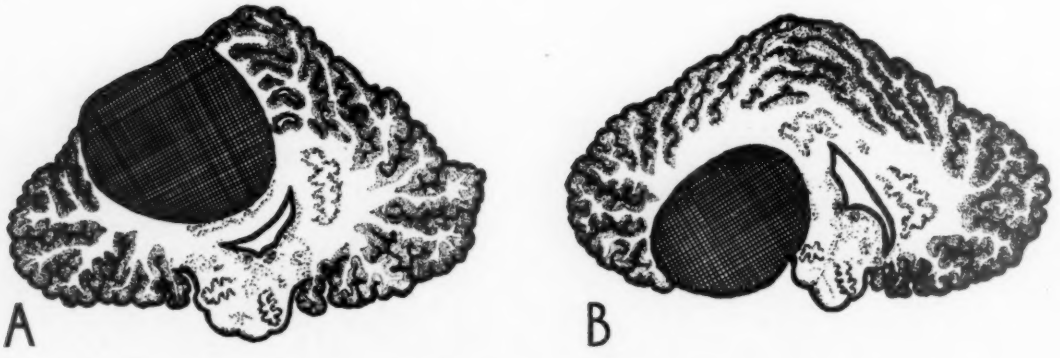

Fig. 7. *A* and *B*.

b. Lateralized Cerebellar Tumors. These tumors always lie lateral to the aqueduct and fourth ventricle; hence, in the sagittal ventriculogram one may expect to find some lateral displacement of the aqueduct and fourth ventricle away from the tumor. Since these lesions may be located either in the ventral or dorsal portion of the cerebellar hemisphere, the displacement of the aqueduct and fourth ventricle, as seen in the lateral view, may simulate the deformity produced by cerebellopontine angle lesions in the first instance, and dorsal vermis lesions in the latter. Drawings of verticofrontal cerebellar sections illustrate the deformities caused by dorsolateral tumors (Fig. 7*A*) and ventrolateral tumors (Fig. 7*B*).

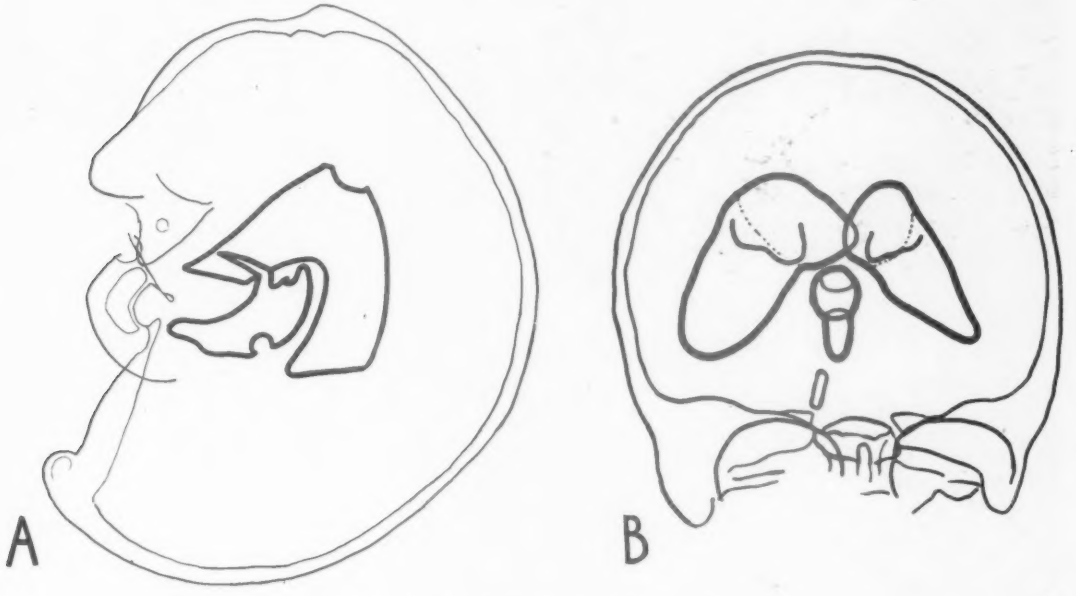

Fig. 8. *A* and *B*. Case III (325991).

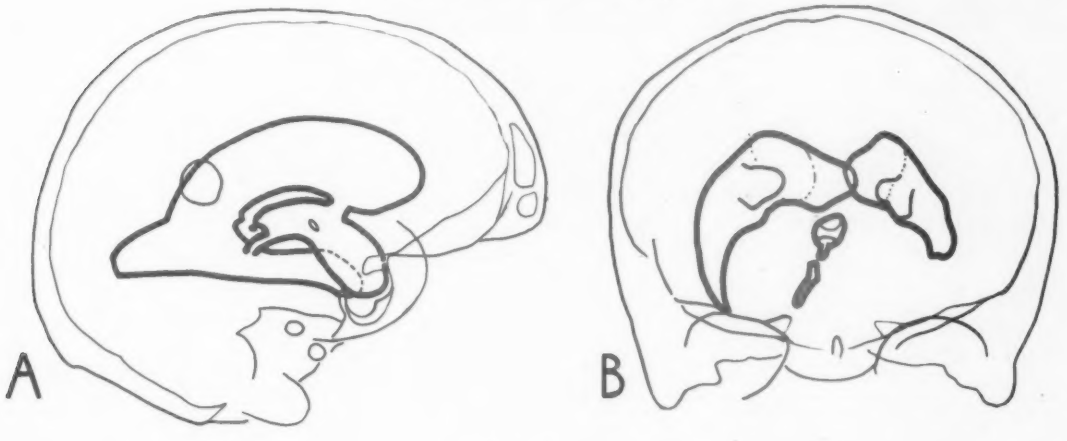

Fig. 9. *A* and *B*. Case IV (256512).

The lesion in Case III was found to be a cystic astrocytoma in the dorsolateral portion of the left cerebellar hemisphere. The lateral ventriculogram, with the occiput superior (Fig. 8*A*), shows the angulation between the aqueduct and the compressed

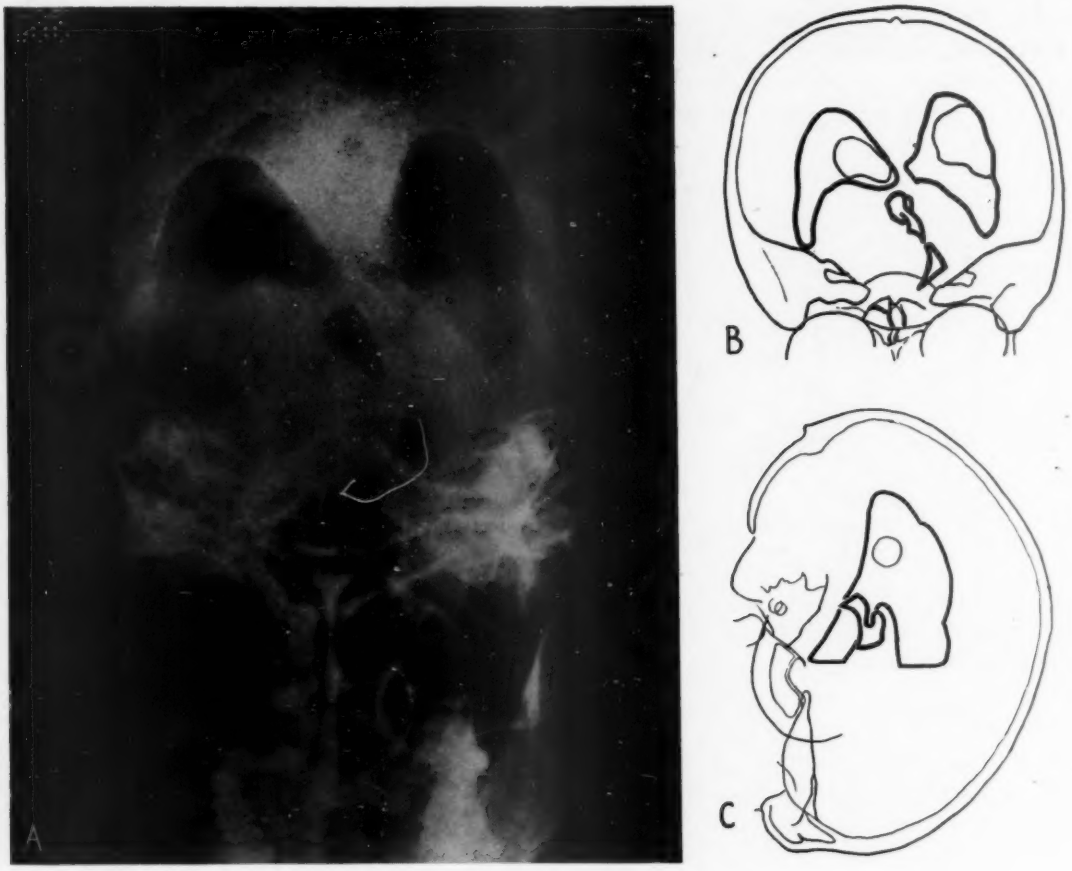

Fig. 10. *A*, *B* and *C*. Case v (381971).

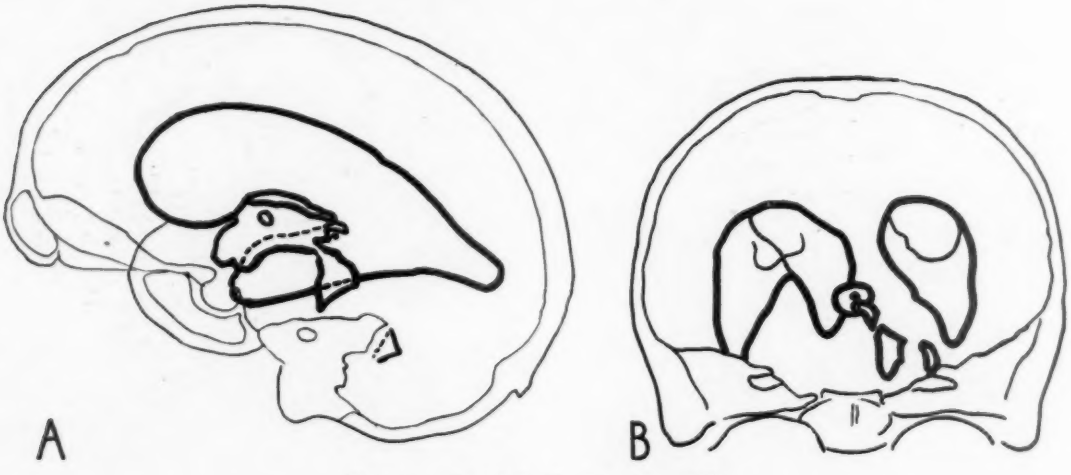

Fig. 11. *A* and *B*. Case vi (373566).

fourth ventricle, simulating the deformity produced by vermis tumors. The occipital sagittal view, on the other hand, provides positive evidence of lateralization by showing contralateral displacement of the partially collapsed fourth ventricle (Fig. 8*B*).

The lateral displacement of the ventricular system caused by cerebellar gliomas is

similar to that produced by subtentorial meningiomas arising from the transverse sinus, since these extracerebellar lesions encroach on the homolateral cerebellar hemisphere. Cases IV and V were meningiomas arising from the left transverse sinus in the first instance, and from the right transverse sinus in the second case. Occipital sagittal ventriculograms (Figs. 9*B*, 10*A* and *B*) demonstrate in both cases displacement of the aqueduct and the compressed fourth ventricle to the side opposite the tumor. In the lateral views (Figs. 9*A* and 10*C*), the fourth ventricle is not visualized, but the aqueduct is seen to be slightly elevated in each instance and the inferior margin of the third ventricle is displaced slightly upward in Case IV. These last mentioned deformities are probably due to displacement of the brain stem by tumor pressure.

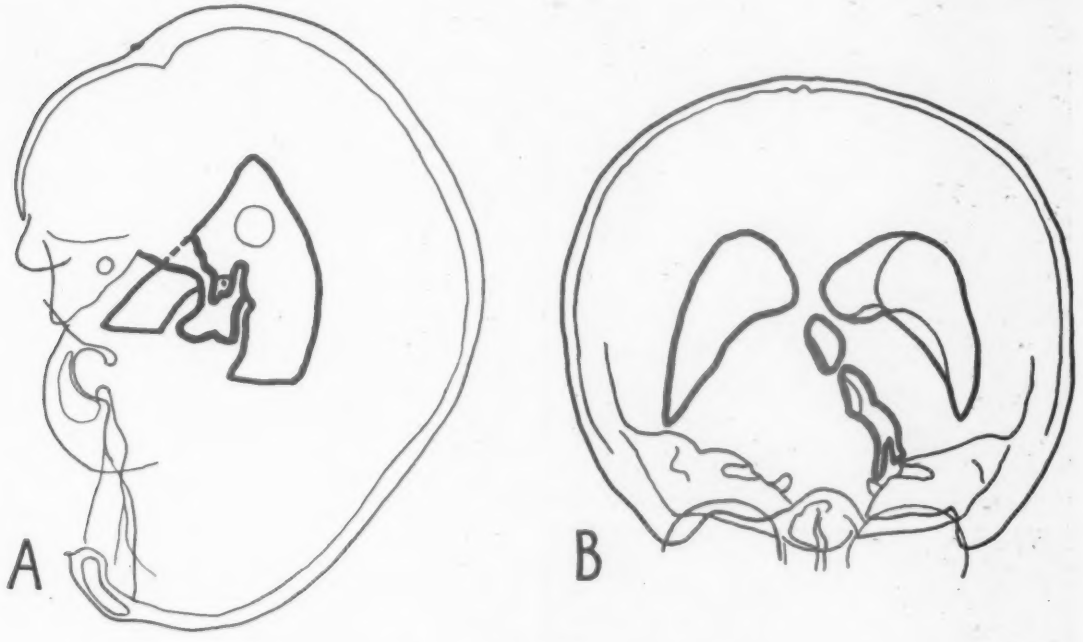

FIG. 12. *A* and *B*. Case VII (380363).

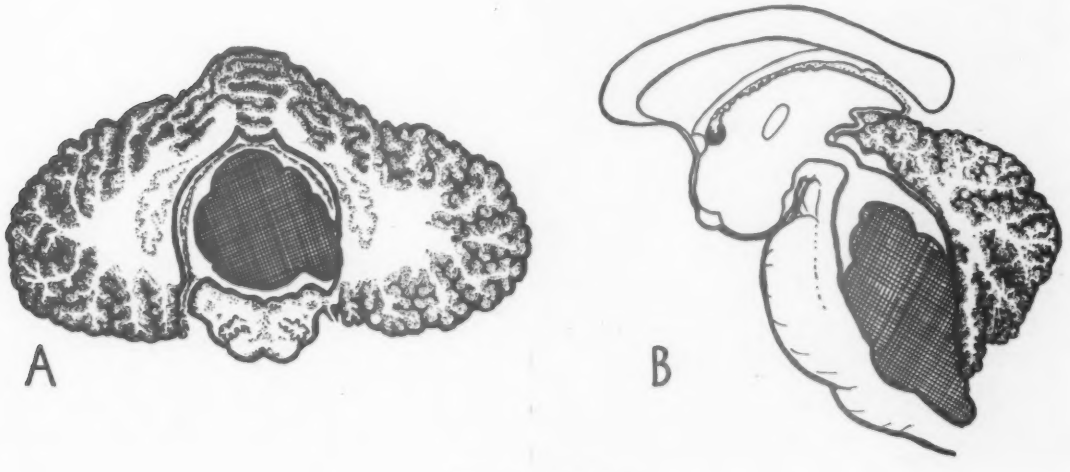

FIG. 13. *A* and *B*.

Ventrolateral cerebellar tumors also cause a considerable ventricular shift to the

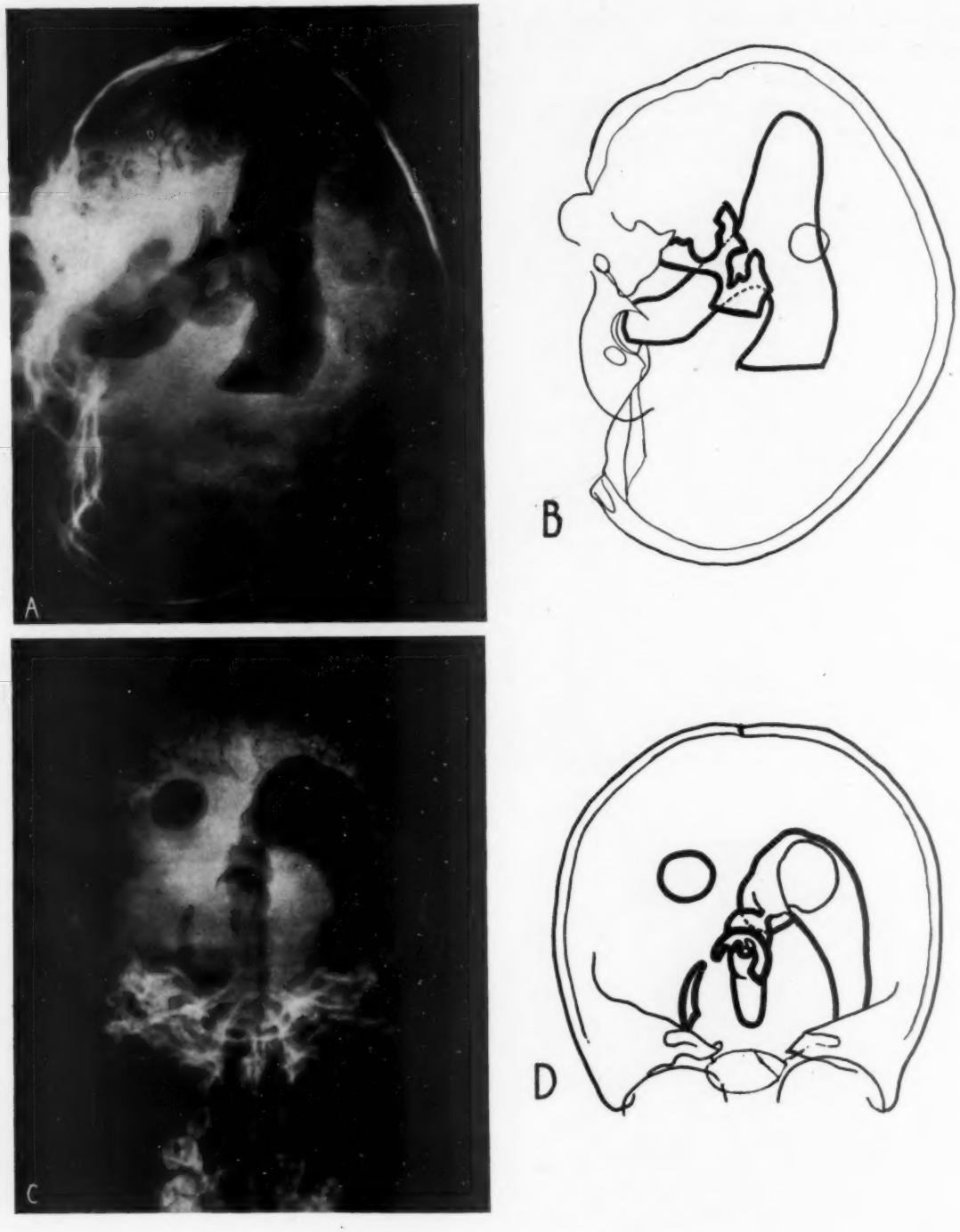

Fig. 14. *A*, *B*, *C* and *D*. Case VIII (343829).

opposite side, or at least a marked filling defect on the tumor side. Tumors in this location may produce some elevation of the aqueduct and fourth ventricle; however, this was not a prominent sign associated with either of the following cases of ventrolateral tumors. A medulloblastoma in Case VI and an astrocytoma in Case VII each occupied the caudal portion of the right cerebellar hemisphere. In the lateral views of

each case (Figs. 11*A* and 12*A*), fairly extensive filling of the fourth ventricle has been accomplished. The sagittal views (Figs. 11*B* and 12*B*) demonstrate a lateral shift of the fourth ventricle and the aqueduct, a finding typical of hemispheral tumors. A localized indentation in the caudal portion of the fourth ventricle on the tumor

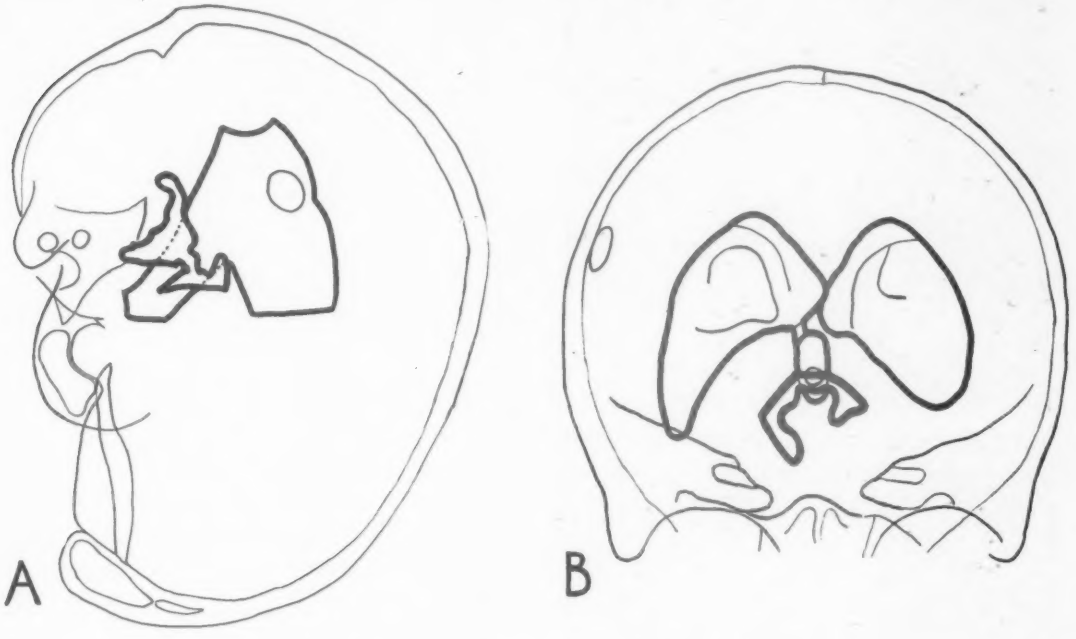

Fig. 15. *A* and *B*. Case ix (351554).

side indicates the inferior position of the tumor.

II. Tumors within the Fourth Ventricle.

These intraventricular neoplasms obstruct the outlet of the fourth ventricle at the foramen of Magendie, thus resulting in dilatation of the entire ventricular system, including the aqueduct and fourth ventricle. The enlargement of the fourth ventricle may be also due to direct distention by the tumor. The occipital sagittal ventriculogram characteristically demonstrates the upper pole of the tumor capped by the dilated dome-shaped proximal portion of the fourth ventricle. Figures 13*A* and *B* aid in explaining the ventriculographic deformity produced by these intraventricular tumors. Figure 13*A* is a schematic drawing of an intraventricular tumor sectioned in a verticofrontal plane, while Figure 13*B* demonstrates the same tumor in the mid sagittal section. If the upper part of the tumor lies free in the cavity of the fourth ventricle, the oxygen may be traced downward along the sides of the tumor into both lateral recesses. However, this distribution of oxygen along the lateral margins is frequently unequal, and such inequality cannot be interpreted as diagnostic of a lateralized attachment of the tumor.

Cases viii and ix illustrate the characteristic appearance of these lesions in lateral and occipital sagittal ventriculograms. At operation the tumor was proved to be an intraventricular ependymoma in Case viii, and a medulloblastoma in Case ix. In the occipital sagittal views of Case viii (Figs. 14*C* and *D*), the right lateral margin of the tumor is outlined by oxygen, while the left lateral margin is not shown, in spite of the fact that there was no attachment on this side. This inequality is also noted in the sagittal view of Case ix (Fig. 15*B*).

The asymmetry of the fourth ventricle produced by the intraventricular astrocy-

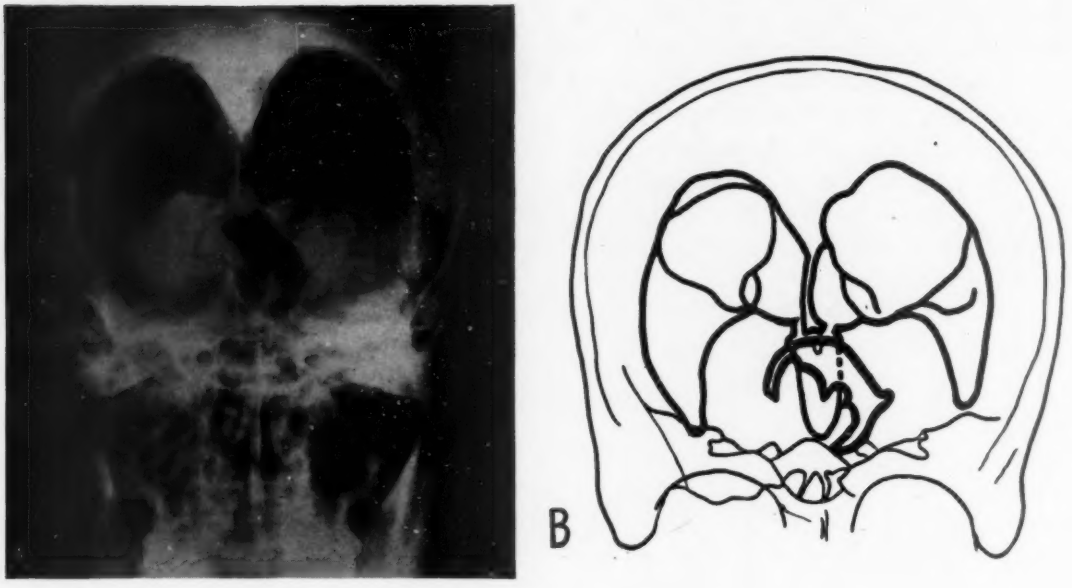

Fig. 16. *A* and *B*. Case x (379055).

toma in Case x (Figs. 16*A* and *B*) suggests wide attachment to the right ventricular wall, or perhaps invasion of the cerebellar

Fig. 17. Case xi (323543).

hemisphere. In this case the sign proved reliable at operation. If oxygen is fairly symmetrically distributed on either side of the tumor, one is justified in assuming midline attachment.

Fourth ventricle tumors are also very well demonstrated in lateral views, particularly in lateral projection with the occiput superior. The upper pole of the tumor divides the dilated fourth ventricle into a dorsal and a ventral recess (Figs. 14*A* and *B*, Case viii and Fig. 15*A*, Case ix). The upper recess outlines the elevated roof of the fourth ventricle and the dorsal surface of the tumor, which may be nodular. A nodular outline of the dorsal surface of a cholesteatoma within the fourth ventricle is well demonstrated in the lateral view of Figure 17*A*, Case xi. The anterior recess is produced by the floor of the fourth ventri cle and the free ventral surface of the tumor. The angulation between the inferior border of the aqueduct and the anterior border of the fourth ventricle, typical of cerebellar tumors, may again be seen here, and is considered indicative of compression with anterior displacement of the pons. It is obvious that only the anterior recess will be inflated if the tumor has a dorsal attachment. Under these circumstances, the roentgen appearance will be similar to that found in a vermis lesion. If, on the other hand, the tumor arises from the floor of the fourth ventricle, only the posterior recess

will be visualized, and then the deformity may simulate that produced by a midline pontine tumor. If the fourth ventricle and aqueduct are completely filled by the tumor, differentiation from other cerebellar tumors becomes impossible. A small tumor obstructing the foramen of Magendie may permit complete visualization of the dilated fourth ventricle without producing any filling defect. The ventriculographic picture may then be indistinguishable from inflammatory obstruction of the foramen of Magendie.

SUMMARY

Tumors of the cerebellum and the fourth ventricle produce such characteristic deformities that their exact location can, as a rule, be determined by technically perfect ventriculographic examination.

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THE INCIDENCE OF HYPEROSTOSIS FRONTALIS INTERNA IN FEMALE PATIENTS ADMITTED TO A MENTAL HOSPITAL*

By W. W. ELDRIDGE, M.D., *Principal Medical Officer, St. Elizabeth's Hospital and Associate in Medicine, George Washington University Medical School and* GEORGE A. HOLM, M.D., *Senior Medical Officer, St. Elizabeth's Hospital*
WASHINGTON, D. C.

INTEREST in a condition called "hyperostosis frontalis interna" was revived by Sherwood Moore[4] in a paper published in September, 1935. In this article Moore gave a comprehensive review of the literature on the subject and yielded to Morel[6] the credit of having introduced the name and of having made the first report of a living example of the condition.

Prior to the publication of Moore's article there was a considerable literature on the anatomy and pathology of the condition, where it was described under various names, but the first clinical description was that of Morel in 1930. Thus, he reintroduced to medical literature a subject which had been a dormant issue for many decades but he described it as one component of a clinical syndrome, since known as Morel's syndrome. This consisted principally of frontal bone hyperostosis associated with obesity, headache, neurological disturbance and a tendency to mental disorder. Since that time there have been numerous reports of individual cases which have usually presented all, or most, of these factors.

Moore's bibliography included reports of studies made on autopsy and museum specimens of varying types of groups, with a total of 65. It also included reports of 3 living cases. The authors quoted were Morgagni,[7] Casati,[1] Stewart,[9] Greig,[2] Morel,[6] van Bogaert,[10] Schiff and Trelles.[8] Moore's personal report was made from the study of the roentgenograms of approximately six thousand skulls of patients from general hospitals and clinics in St. Louis, Missouri.

In this large group he found 72 skulls which showed the presence of a hyperostosis of the inner table of the frontal bone, an incidence of 1.2 per cent. In a later article[5] published in 1936, Moore reported the examination of the skulls of 695 additional patients among whom was a group of 33 females suffering with psychoses. He found 24 additional examples of localized frontal hyperostosis in this second study, 14 of which occurred in the psychotic group. This gave an incidence of 1.41 per cent for the combined groups of the two studies.

In his 1936 report Moore also described several other more or less similar conditions of the skull. The whole communication was offered under a title which signified that all of these conditions are manifestations of a metabolic craniopathy. He stated that the surest method of making a diagnosis of any of these conditions was by means of roentgenographic examination.

It seemed to us that the most striking feature of Moore's later report was the fact that in a group of 33 psychotic females he found the condition in 14 instances, which was a strikingly high incidence, 42.4 per cent, as compared with the low incidence of 1.22 per cent in his original communication based on 6,000 cases taken from the general population. We wondered at his lack of comment on this feature of the situation. We were led to believe that this curious circumstance was worthy of further investigation.

In view of this and of the many references in the literature to psychotic and neurologic manifestations in these patients, we decided to carry out routine roentgenographic examinations of the skulls of female patients consecutively admitted to St. Elizabeth's Hospital, an institution for the care and treatment of mental disease.

* Read before the Mid-Winter Conference of Eastern Radiologists, Washington, D. C., Feb. 10–11, 1939.

In carrying out this plan no selection of patients was made and the group included both white and colored females ranging in age from twenty-three to eighty-seven years, drawn from the civilian population of the District of Columbia. In each instance lateral and posteroanterior views were taken and in many instances the films were made stereoscopically. The group finally studied consisted of 200 patients.

We accepted the description of Moore as the criterion by which to judge the presence of the condition roentgenographically, and a group of four of us examined all films together, no case being accepted unless a positive diagnosis was concurred in unanimously. In the group of 200 cases examined 50 unquestionably presented the condition here discussed. This is an incidence of 25 per cent. The accompanying illustrations show reproductions of typical films.

The next step in the investigation was to examine the histories and reports of both physical and mental examinations in each of the positive cases. In each instance the following factors were given special attention: age, color, mental diagnosis, serology, menstrual history, marital status, pregnancies, headaches, visual defects, obesity and neurological disorders.

Fig. 1. Posteroanterior view of skull showing typical nodular form of hyperostosis frontalis interna.

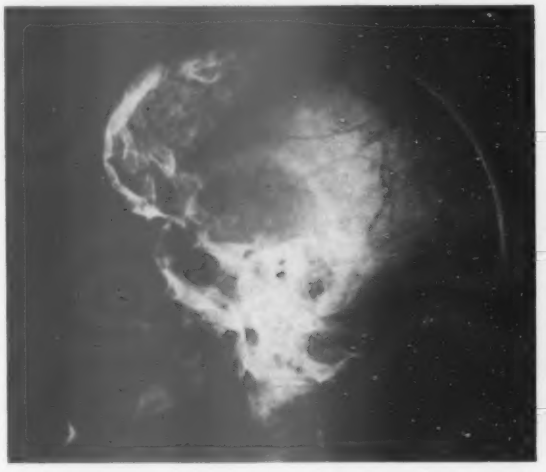

Fig. 2. Lateral view of skull of typical case of nodular form of hyperostosis frontalis interna.

The ratio of white to colored women was found to be approximately the same as that in the total hospital female population and there was no correlation between the presence of the hyperostosis and the menstrual history. There were 35 married and 15 single women. The former had a history of 72 pregnancies, or about 2 per individual. The incidence in age groups showed the greatest number to have been in the fifth decade of life. Table 1 shows the incidence in the age groups by decades.

Table I

INCIDENCE OF HYPEROSTOSIS FRONTALIS INTERNA IN AGE GROUPS, BY DECADES

Age	Number of Positive Cases
20–29	6
30–39	8
40–49	10
50–59	9
60–69	9
70–79	4
80–89	4
	50

FIG. 3. Lateral view of skull showing a modified form of senile type of hyperostosis frontalis interna.

It appears that although 72 per cent of the cases occurred after the fortieth year of life, nevertheless, contrary to the impression gained from the literature, a considerable proportion, 28 per cent, of the cases occurred in the earlier years. Ten per cent of the cases had a positive blood Kolmer relaction, which is approximately the same ratio as that found in the general mental hospital population, and presumably had no bearing on the situation.

TABLE II

INCIDENCE RELATIONSHIP OF MENTAL DIAGNOSIS TO HYPEROSTOSIS FRONTALIS INTERNA

Mental Diagnosis	Number of Positive Cases
Dementia precox, hebephrenic	11
Dementia precox, catatonic	4
Dementia precox, paranoid	5
Psychosis with cereb. arteriosclerosis	11
Senile psychosis	4
Involutional psychosis	2
Manic depressive psychosis	5
Paranoid state	2
Psychosis with somatic disease	1
Psychosis with syphilitic meningo-encephalitis (paresis)	2
Epilepsy	1
Mental deficiency	1
Undiagnosed psychosis	1
	50

In reviewing the cases in a search for the factors usually alleged to constitute the syndrome, namely, obesity, headache, visual defect and neurological disorder, we found that obesity occurred in only 8 cases, or 15 per cent, and in none of these was the obesity of a type suggesting a definite endocrine disorder. Visual defects, except those due to trauma or senescence, were not present in any case. Headache was a basis of complaint in only 3 cases and neurological disorders, except those associated with epilepsy and paresis, were absent in all but one case and this case exhibited impaired hearing only.

In the light of the above observations it is impossible for us to concur in the opinion that a definite syndrome, sometimes referred to as the "triad of Morel," is present in cases of localized hyperostosis of the frontal bone, at least so far as the female population of a mental hospital is concerned.

A study of the incidence relationship to the mental diagnoses of these 50 cases is summarized in Table II. It will be seen that

FIG. 4. Specimen from autopsy showing localized thickening of inner table of frontal bone with obliteration of intertabular space.

of the twenty-two types of psychoses classified by the American Psychiatric Association, thirteen are represented in this group of cases. These particular types are those of most frequent occurrence and constitute the majority of all cases of psychoses. Furthermore, it is of interest to note the fact that the largest number of cases in this study occur in two types of psychoses which usually predominate in a mental hospital, namely the dementia precox and the senescent-arteriosclerotic groups.

It therefore appears obvious that the occurrence of hyperostosis frontalis interna does not have any close or significant relationship to any one particular mental disorder.

Many hypotheses have been suggested as bearing on the etiology of frontal bone hyperostosis. Greig[2] in 1928 offered the suggestion that disuse absorption of calcium from other parts of the body in excess of the excretory powers resulted in the excess being deposited in the frontal region as an innocuous relief of a system saturated with calcium, an area which age renders least important. Moore[5] more recently advanced the idea that it is the result of some metabolic disturbance bearing chiefly on a special calcium metabolism in the female. Henschen[3] considered it merely a very frequent tertiary sex characteristic.

We have nothing to add to these speculations in regard to etiology but we believe that the condition is one which merits further extensive investigation, in both the general population and in the special population of mental hospitals. To us the most significant fact observed in our group of cases is that the localized condition called hyperostosis frontalis interna occurs about twenty times as frequently in females admitted, with mental disease, to a mental hospital as it occurs in the general population.

We therefore feel justified in regarding it as a broad, general, common factor occurring in many types of mental disease. Of what significance this is and whether or not it is related in any way to etiological factors of mental disease is a question of considerable interest to us but one for which we have no answer.

SUMMARY

A roentgenographic study was made of the skulls of 200 female patients consecutively admitted, with mental disease, to a mental hospital.

In this group, 50 cases, or 25 per cent, were found to present localized thickening and increased density of the inner table of the frontal bone, a condition termed hyperostosis frontalis interna.

It is a noteworthy fact that this incidence is twenty times as great as that found in the general population.

Clinical examination and personal history of our cases did not show any occurrence of the syndrome described by Morel and noted by many other authors.

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PERSISTENT FIBRIN BODY

A PROBLEM IN DIAGNOSIS*

By ARTHUR REST, B.Sc., M.D.
Acting Medical Director
SPIVAK, COLORADO

ALTHOUGH pleural effusions are common accompaniments of artificial pneumothorax, a residual fibrinous mass, or "fibrin body," occurs but rarely. Sachs[1] found 2 occurrences in 700 cases of pneumothorax. Hager and Langebeckmann[2] discovered only 1 in 400 cases. Mende[3] found 3 in 512 cases. Zavod[4] reported 5 cases, 1 of which was confirmed by autopsy. Out of 328 cases at the Sanatorium of the Jewish Consumptives' Relief Society, 1 developed a fibrin body during residence, and 3 were admitted with this sequel.

When this residuum follows a hydropneumothorax, it is usually produced by a massive serous, or more likely, a sanguineous effusion, although this is not the rule. Even a small effusion may result in this formation. The sanguineous type may be more conducive to fibrin body formation in that the fibrin content is greater. The "fibrin body" has certain characteristics and can easily be identified, but only by roentgenological examination. It has a spheroidal configuration of variable size, generally rests on the diaphragm, and is usually freely movable in the pleural space when pneumothorax is present (Fig. 1, *A* and *B*). The mass may increase in diameter by additions of fibrin from subsequent effusions. On the contrary, it has on occasion been known to disappear after an effusion. Usually, with the re-expansion of the lung, these foreign bodies are in some way absorbed and disappear completely. Zavod, however, states that they may persist roentgenologically for a period of four weeks to four years. As a rule, they produce no symptoms, but in the case reported there was a constant dull ache over the left lateral chest wall, apparently due to pressure.

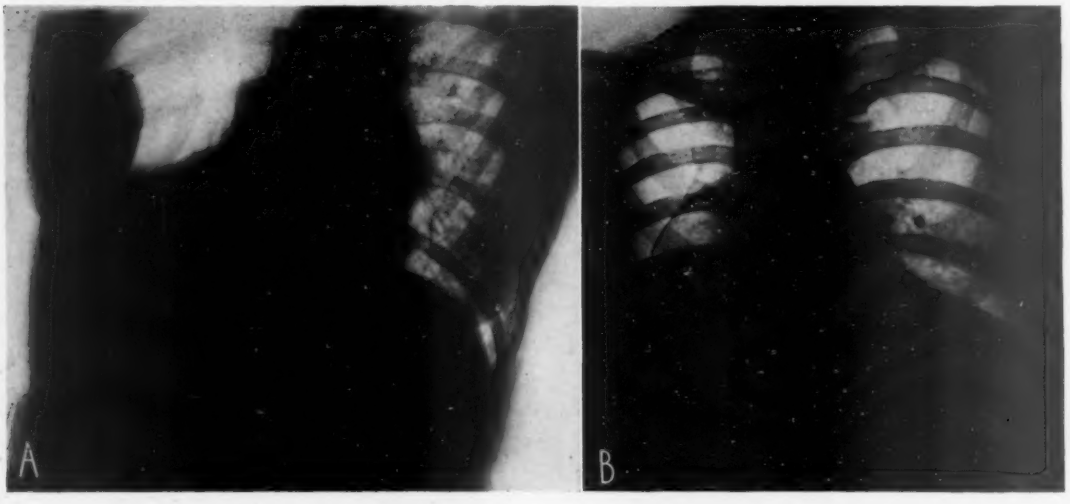

FIG. 1. *A*, fibrin body, ovoid in shape, resting in the right pneumothorax cavity at the costophrenic angle. Right lung is almost completely collapsed. *B*, same fibrin body. The right lung is re-expanding.

The case presented was intriguing because the persistence of the fibrin body after re-expansion of the lung led to a

* From the Medical Department of the Sanatorium of the Jewish Consumptives' Relief Society, Spivak, Colorado.

roentgen-ray configuration of considerable diagnostic interest.

REPORT OF CASE

L. W., female, aged thirty-nine, was admitted to the Laurel Beach Sanatorium at Seattle, Washington, on April 3, 1937. The onset of pulmonary tuberculosis was pleuritic in 1935. On entrance to that institution cough, expectoration, weakness and streaking were found to be the cardinal symptoms. Examination there revealed a moderately advanced

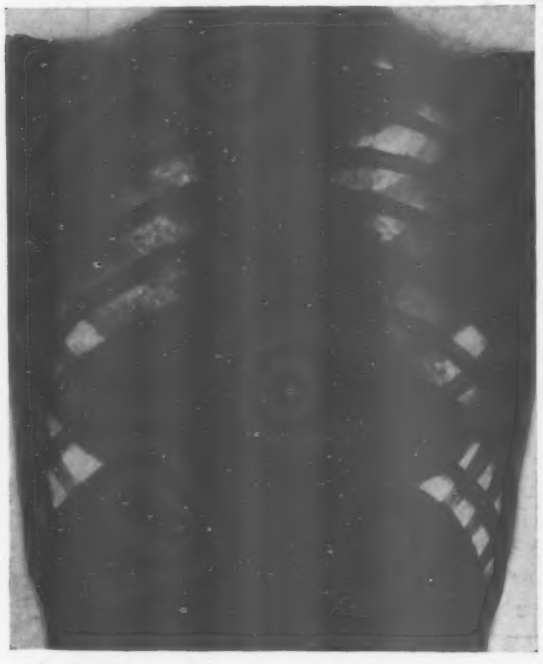

Fig. 2. Before pneumothorax—a moderately advanced bilateral infiltrative lesion.

infiltrative lesion involving both lungs (Fig. 2). Sputum was positive. A left artificial pneumothorax was instituted on May 15, 1937, because of hemoptysis, and discontinued six weeks later on account of adhesions (Fig. 3) and a complicating effusion (Fig. 4). Patient left on December 12, 1937.

She was admitted to the Sanatorium of the Jewish Consumptives' Relief Society on December 14, 1938, in an extremely emaciated condition. Cough was severe, and was daily productive of 2 ounces of sputum, which was positive. Her weight of 77 pounds was 55 pounds below standard. Examination revealed a small exudative lesion in the right upper lobe,

Fig. 3. Left artificial pneumothorax. Lung between the second and fifth costal cartilages is adherent to the lateral chest wall. Small amount of fluid in the costophrenic angle.

with probable cavitation in the apex. In addition she had findings of increased left cardiac dullness and dullness at the left base. Roent-

Fig. 4. Shows a left hydropheumothorax; formation of the fibrin body was preceded by an effusion.

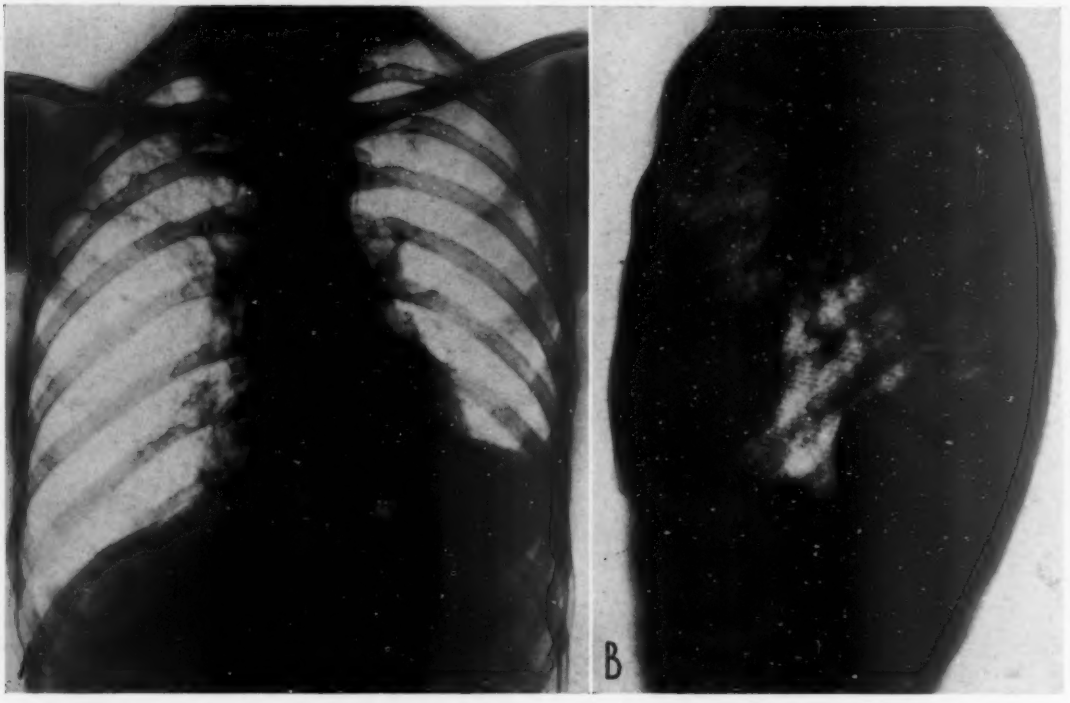

Fig. 5. *A*, exudative lesion of the right upper lobe with probable cavitation. There is a spheriodal mass about 3 inches in diameter, situated off the left heart border and resting on the diaphragm. The mass is sharply outlined and uniformly opaque. *B*, lateral view of fibrin body apparently attached to the left lateral chest wall.

Fig. 6. Roentgenogram of March 21, 1939; almost complete disappearance of the fibrin body.

genologically there was a spherical mass about 3 inches in diameter situated on the left diaphragm, posteriorly to the breast. The mass was sharply outlined, apparently attached to the left lateral chest wall, and uniformly opaque (Fig. 5, *A* and *B*).

A problem in diagnosis with regard to the nature of the mass presented itself. Fibrin body, calcified lung abscess, xanthoma, adenocarcinoma of the lung, dermoid cyst, and echinococcus cyst were among the various considerations. A left artificial pneumothorax was attempted as a diagnostic measure but there was no free pleural space.

Subsequently, the previous history and roentgenograms were obtained from the Laurel Beach Sanatorium, Seattle. These revealed that the mass had not been present prior to the induction of pneumothorax (Fig. 2) and that its evolution was preceded by an effusion (Fig. 4). It could now be interpreted as a persisting fibrin body (Fig. 5, *A* and *B*). The last roentgenogram of March 21, 1939 (Fig. 6) shows the fibrin body almost completely absorbed.

CONCLUSIONS

1. A fibrin body may occur as a harmless sequel of hydropneumothorax and can be diagnosed only by roentgen examination.

2. A case is reported of a pleural foreign body persisting two years after the discontinuation of pneumothorax; the roentgenological appearance of the mass without a coexisting pneumothorax space led to an interesting diagnostic problem.

3. The supposition that these fibrin bodies disappear promptly with the re-expansion of the lung is erroneous and might lead to false conclusions.

I am indebted to Dr. Frederick Slyfield of the Laurel Beach Sanatorium, Seattle, Washington, for his kindness in submitting to me the valuable roentgenograms in the above case (Figs. 2, 3 and 4).

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CARCINOMA AS A COMPLICATION OF ACHALASIA OF THE CARDIA*

By KARL KORNBLUM, M.D., *and* LAWRENCE C. FISHER, M.D.
PHILADELPHIA, PENNSYLVANIA

ONE of the most interesting and fascinating lesions of the alimentary tract is the condition commonly referred to as cardiospasm; interesting because of its controversial etiology, fascinating because of its spectacular clinical and roentgenological manifestations. For this lesion Walton has given the following definition: "A condition of dilatation and hypertrophy of the esophagus where, on post-mortem examination, no obstruction can be found distal to the dilatation."

When writing on this subject one is immediately confronted with the problem of a name. It has been referred to by so many names that there is now a long list of synonyms, such as idiopathic dilatation of the esophagus, hiatal esophagismus, primary dilatation of the esophagus, esophagectasia, cardiospasm, idiopathic hypertrophy of the esophagus, phrenospasm, preventriculosis and achalasia of the cardia. In this country the condition is most popularly known as cardiospasm, given preference no doubt because of its brevity as well as a misconception regarding its etiology. However, to one not familiar with this affection the name cardiospasm is misleading since it has been amply demonstrated by many investigators that there is no actual spasm of the cardiac orifice of the stomach. Furthermore, the term would seem undesirable since a true cardiospasm does occur but does not give rise to the pathological changes found in the condition under discussion. It seems reasonable to suppose that the cardiac orifice may become spastic as the result of local irritation or reflexly from pathological changes at a distance. Thus it is believed that fissures, ulcers or inflammatory changes in the lower end of the esophagus or fundus of the stomach may result in spasm of the cardia as occurs at the pylorus, the ileocecal region or anal sphincter. Likewise, it is thought that gross intra-abdominal lesions such as gallbladder disease may reflexly produce spasm of the cardia. Thus, it would seem necessary to differentiate between a true cardiospasm and the condition which we prefer to call achalasia of the cardia.

In using the term achalasia of the cardia we have chosen to follow the concept of Hurst regarding the pathogenesis of this condition. This concept has its inception in the physiological investigations of Kronecker and Meltzer who in 1883 showed that "every act of swallowing easily opens the cardia by reflex action." Accordingly, Einhorn, in 1888, describing a case of marked dilatation of the esophagus, concluded that it was due to "a lack in the reflex relaxation or opening of the cardia during the act of swallowing." This theory was lost sight of until Rolleston in 1896 made a similar suggestion. However, again this idea was given slight attention until Hurst, in 1913, quite independently brought forth the same theory. On the suggestion of Sir Cooper Perry the term "achalasia" meaning "absence of relaxation" was introduced by Hurst in 1915.

While this concept gives us a name and a mechanism of production of the changes as observed clinically it still leaves unsolved the matter of etiology. Believing that the primary affection responsible for the condition was to be found in the nervous system, Hurst in 1924 suggested that "the majority of cases . . . are caused by progressive organic disease involving Auerbach's plexus." This nerve plexus had been more or less neglected in previous studies of this condition. In early cases of achalasia

* From the Departments of Radiology of the Jefferson Medical College Hospital and the Graduate Hospital of the University of Pennsylvania, Philadelphia, Pa. Read at the Thirty-ninth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 19–22, 1938.

of the cardia there is only a compensatory hypertrophy of the esophagus, while dilatation occurs only after compensation fails. Studying these early cases pathologically, Rake found lesions of a subacute inflammatory nature involving Auerbach's plexus, the changes being most marked at the cardia. In advanced cases with dilatation, the plexus and surrounding tissues were converted into a dense fibrous scar with a complete disappearance of the ganglion cells. These changes he believed are responsible for the failure of relaxation of the cardia although what initiates the alterations in the nerve plexus is apparently still unknown. The finding of inflammatory changes in Auerbach's plexus suggests that the lesion is an acquired one, yet there is much to lead one to suspect, as several writers have suggested, that it is of congenital origin.

Jackson would place the blame for this condition on the diaphragm, describing a diaphragmatic pinchcock, along with kinking of the abdominal esophagus as the normal mechanism by which food in the stomach is prevented from regurgitating back into the esophagus. Accordingly it is the failure of the diaphragmatic pinchcock to open normally that constitutes the stenosis in so-called cardiospasm.

It is not the purpose of this communication to deal with the etiology of this condition. Among the numerous theories that have been proposed only those of Hurst and Jackson have been mentioned since the concept of an achalasia appeals to us as being the most probable immediate cause of the condition. There most certainly seems to be an "absence of relaxation" of something or other, whether of the cardia or the diaphragmatic pinchcock we are in no position to say, although we rather favor Hurst's idea that the abdominal esophagus and a short section above the diaphragm is a true sphincter. This would harmonize with our own frequent roentgenological observation that the obstruction in achalasia of the cardia is sometimes above and sometimes below the diaphragm.

Before proceeding with our justification for this presentation we are prompted by custom and habit to give the "firsts" of the subject. Most of the present-day literature mentions Purton, 1821, as the first to describe a case of dilatation of the esophagus without obstruction, although Hannay who published an account of a case in 1833 is generally credited with having been first to recognize the condition. This would make our acquaintance with achalasia of the cardia of fairly recent origin which would be strange indeed considering the very striking clinical manifestations of this condition. It seems more probable that the affection, as a clinical entity, was known even in the earliest days of Medicine. We are indebted to Dr. H. I. Goldstein who has called our attention to a recorded account of the condition which dates back to 1679 when Thomas Willis published his Pharmaceutice Rationalis in which he describes a case of persistent vomiting, the clinical manifestations being typically those of achalasia of the cardia. Willis kept his patient alive and well for at least fifteen years by means of an instrument he devised for passage through the esophagus into the stomach, described as "a Rod, of a whale Bone, with a little round Button of Sponge fixed to the top of it." An improvised bougie which served an excellent purpose. The concluding remark of Willis is of interest in connection with the present consideration of carcinoma as a complication of achalasia of the cardia. To quote: "Without doubt in this case the Mouth of the Stomach being always closed, either by Tumour or Palsie, nothing could be admitted into the Ventricle unless it were violently opened." This very interesting account is to be found in Major's "Classic Descriptions of Disease." Returning to more recent activities we find that in 1895 Rosenheim first examined a case with the esophagoscope and in 1897 Rumpel first used the roentgen ray in studying this condition.

We are also tempted to mention certain facts of general interest with which we were unfamiliar before making the present inves-

tigation, although they are not directly related to the matter at hand. Regarding the sex incidence we find that the condition is somewhat more frequent in men than in women. This does not coincide with our own somewhat limited experience. Furthermore, the affection is not peculiar to young neurotic females and has nothing whatever to do with psychoneurosis. Also of interest is the fact that salivation is a common complaint and results in considerable hypertrophy of the salivary glands.

Judging from reported cases, carcinoma as a complication of achalasia of the cardia is of rather uncommon occurrence, although Walton mentions Gottstein as stating that unless cured by treatment this complication frequently develops. In Gottstein's series of 33 cases there were 3 carcinomas. It is of interest that in one of the earliest cases to be reported there was an epithelioma. This case reported by Fagge was a man who, dying at the age of eighty-four, had complained of difficulty in swallowing for forty years. His symptoms were characteristic of achalasia of the cardia. At postmortem examination the only abnormal findings were in the esophagus which contained an epithelioma near its lower end. Apparently this was the cause of the patient's death. Below the growth the esophagus, for several inches extending down to and including the cardia, was markedly narrowed. The rest of the esophagus was considerably dilated and hypertrophied. Fagge considered the lesion a congenital stenosis of the lower end of the esophagus and this phase of the case interested him more than did the epithelioma occurring as a complication, as well it might.

Plummer, reporting a series of 301 cases, lists as associated conditions syphilis and psychoneurosis but makes no mention of carcinoma. One of his patients, however, died of a carcinoma of the esophagus eight months after treatment for achalasia of the cardia. Plummer states that in this case the diagnosis of achalasia may be questioned but the patient had had symptoms for five to six years which along with the roentgenological manifestations were characteristic of achalasia. Furthermore, after dilatation the patient was relieved of her dysphagia and had no further difficulty when last heard from which was two months before her death.

Vinson, reporting on the treatment of 683 cases of achalasia of the cardia, makes no mention of carcinoma as a complication. A report dealing specifically with epithelioma in association with achalasia of the cardia was made in 1931 by Rake who found 3 malignancies in 15 pathological specimens of this condition examined by him. He discusses the pathology of the condition and the basis for the development of carcinoma.

When one inquires into the pathology of achalasia of the cardia it is somewhat surprising that cancer as a complication is not of more frequent occurrence. It is no doubt true that some of these cases are being overlooked, but with the increasing frequency of postmortem examinations and the more universal employment of the esophagoscope it seems unlikely that any large number of cases are being missed. While we have not tabulated the actual number of cases of achalasia of the cardia that we have examined roentgenologically our experience no doubt is similar to that of hundreds of other roentgenologists working in a large city hospital. During this experience we have encountered 2 cases of carcinoma in association with achalasia of the cardia, and these form the basis of this report.

Significant in the pathology of uncomplicated cases of achalasia of the cardia is the absence of any organic obstruction at the lower end of the esophagus and yet roentgenologically the evidence of obstruction is the outstanding manifestation. As the result of the functional obstruction produced by failure of relaxation there occurs a state of hyperperistalsis of the thoracic esophagus whenever food or liquids are ingested. In consequence, over a period of years the thoracic esophagus shows evi-

dence of muscular hypertrophy, a compensatory mechanism. As long as this compensation is maintained no symptoms are produced, the patient is unaware of the condition, and in this early stage the lesion apparently cannot be diagnosed clinically. Whether any roentgenological manifestations are present at this stage of the disease that would enable one to recognize the condition is questionable. After muscular hypertrophy has reached a maximum and compensation begins to fail, dilatation of the esophagus occurs and this increases throughout the subsequent course of the disease unless checked by appropriate treatment. Eventually the entire thoracic esophagus becomes dilated and with this extreme involvement it becomes elongated and tortuous. Apparently the cervical esophagus never becomes much involved. We have on repeated occasions attempted to completely fill the esophagus with an opaque medium up to the hypopharynx but never with success. According to Hurst, when the esophageal contents form a column 8 inches high the cardia opens, permitting some of the material to enter the stomach. Presumably the weight of the contents is then sufficient to overcome the obstruction offered by the cardia. The patient soon learns the necessity of taking in a certain amount of food or liquids in order to have any enter the stomach.

With the onset of dilatation the beginning of stagnation of esophageal contents occurs. This is at first intermittent but with progression of the disease there comes a time when the esophagus is never completely empty. The onset of stagnation initiates the period of secondary changes in the esophageal wall. Acting as an irritant the decomposing food produces inflammatory changes in the esophageal mucosa, giving rise to an esophagitis. The mucosa becomes flattened and smooth and subsequently ulcerated. The submucosa becomes greatly thickened but fibrosis with constriction apparently does not occur. These inflammatory changes may give rise to a true cardiospasm which is thus superimposed upon the achalasia. No doubt this is responsible for the conflicting statements that appear in the literature regarding the nature of the obstruction at the cardia and is probably also responsible for the name of cardiospasm being applied to this condition.

The ulcers which occur secondarily are usually shallow in character but occasionally they may penetrate with subsequent perforation of the esophagus. Ulceration may become so extensive that eventually nearly the entire mucosa may be destroyed. Numerous small islands of epithelium usually remain, however, and these under constant irritation from the decomposing esophageal contents undergo a hyperplasia resulting in small wart-like nodules or papillomata. These may subsequently become malignant and the occurrence of carcinoma in association with achalasia of the cardia no doubt is on this basis. The extent of the secondary changes is dependent upon the height of the stagnant esophageal contents. Since these secondary changes are apparently an invariable accompaniment of the advanced stages of the disease it is indeed surprising that malignant change is not more frequently encountered.

The diagnosis of achalasia of the cardia is in the majority of cases relatively simple. Since symptoms do not appear until dilatation of the esophagus occurs and since this dilatation may be expected to proceed rather slowly one may anticipate that the onset of the clinical manifestations will be insidious. When the patient's complaints have become sufficient to cause him to seek medical attention one may be assured that the disease is of some duration and well established. The history, therefore, is usually one of increasing difficulty in swallowing over a period of years although occasionally the onset is quite abrupt. Dysphagia is intermittent at first but subsequently becomes more or less constant. While this is the outstanding symptom there are others which serve to produce a definite clinical picture. Regurgitation is a fairly constant one. Actual vomiting does not occur and

there is no nausea or retching. The regurgitated material contains unchanged food, much mucus and no free hydrochloric acid. Substernal pain is often present and may at times be the predominant complaint. Certain respiratory symptoms such as cough and dyspnea after eating may be complained of. Excessive salivation may be a distressing symptom. This combination of clinical manifestations are such as to lead one to suspect the existence of an achalasia of the cardia, but the diagnosis should be established by a roentgenologic examination, the passage of bougies and direct visualization through the esophagoscope.

Roentgenologically the typical case offers no diagnostic difficulties. When observed roentgenoscopically the obvious obstruction either at the level of the diaphragm or at the cardiac orifice with the marked dilatation of the thoracic esophagus produces a picture unlike anything else seen in the esophagus (Figs. 3 and 4). The lower esophagus ends abruptly presenting a smooth funnel-shaped contour or at times may be somewhat angulated. There is no irregular filling defect as seen in cancer. The dilatation varies considerably in different cases but is usually much greater than is encountered in any other type of obstructive lesion of the esophagus. When marked, there is noted considerable tortuosity which causes the esophagus to bulge to the right beyond the cardiac silhouette (Fig. 1). Usually the obstruction is complete so that none of the contents enters the stomach until the amount in the esophagus reaches a certain height when a thin stream of the opaque material will be seen entering the stomach. This feature is quite striking and is not to be observed in other esophageal lesions. The characteristic manner in which the opaque medium falls through the esophagus indicates the presence of a large amount of fluid and the mottled appearance presented by the dilated esophageal shadow is due to the presence of retained food. A distinct fluid level is often discernible located high in the thorax or in the lower part of the neck. In the lateral view the enlarged esophagus tends to displace the trachea forward. This along with a fluid level may lead one to suspect the condition on the plain films of the chest (Fig. 2). This appearance is not, however, pathognomonic of achalasia since it is also seen in carcinoma of the esophagus, diverticulum and benign stenosis. In the lower part of the chest just above the diaphragm there will be noted a triangular shadow, apex directed upward. This is due to the shadow of the dilated fluid-filled esophagus superimposed upon the cardiac shadow. This appearance along with the displaced trachea and fluid level will probably not be found in any other esophageal condition.

Plummer warns against accepting the diagnosis of so-called cardiospasm on evidence presented by roentgenographic examination without considering all features of the case, and with this warning we are in hearty agreement. We believe the investigation of a case of achalasia of the cardia is not complete without a careful esophagoscopic examination. This is contrary to the statement made by Plummer in 1921 when he says that "an esophagoscopic examination in the majority of cases is quite unnecessary, knowledge obtained is usually not worth the effort," while Hurst and Rake writing in 1930 state that, "esophagoscopy is superfluous, as the diagnosis can be made with more certainty and much less unpleasantness by means of the x-rays." These are probably not the present-day views of authorities on this subject. Rake, discussing epithelioma in association with achalasia of the cardia in 1931 states that "frequent esophagoscopic examinations should be done to detect such changes early." The cases here reported testify as to the value of the esophagoscopic examination. There is no other way to detect the secondary changes occurring in the esophagus and certainly a knowledge of such changes is important in the proper management of a case of achalasia of the cardia.

The following case is quite characteristic

both clinically and roentgenologically of the typical case of achalasia of the cardia.

Case 1. E. K., white female, aged fifty-five. This patient's chief complaint was vomiting in the evening when she lay down, accompanied by choking sensation. This symptom was more of a regurgitation than a true vomiting and she had complained of this intermittently for the past thirteen years. The material regurgitated was the food as it was ingested. There was considerable mucus present but never any blood. No pain but occasionally she complained of a sense of fullness beneath the sternum. Her food would seem to lodge somewhere in the chest. She has gradually lost weight since the onset of her symptoms and now she is quite weak. She appears fairly well nourished, however. She has been under the care of many physicians during the past thirteen years who have made various diagnoses but none have ever suggested or requested a roentgen examination.

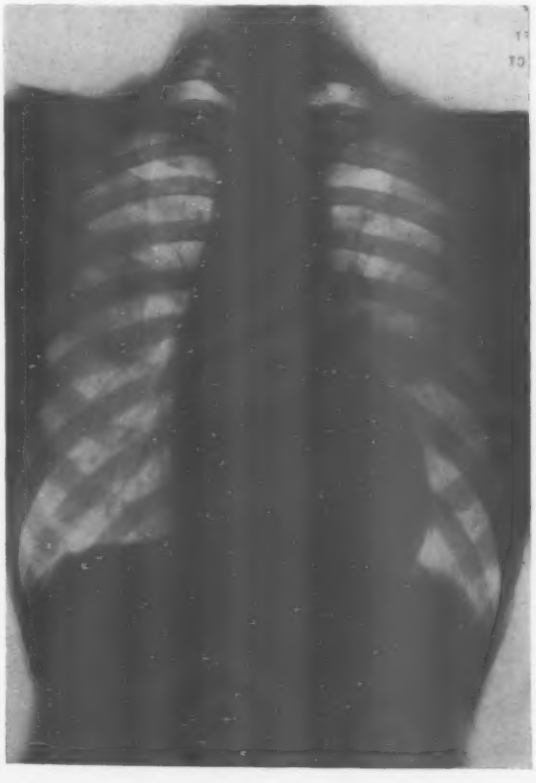

Fig. 1. Case 1. Chest in a case of achalasia of the cardia. What appears to be an enlargement of the heart to the right is actually the markedly dilated esophagus extending to the right as seen in Figure 4.

Fig. 2. Case 1. Lateral view of chest. Note forward displacement of trachea and fluid level, also triangular shadow just above the diaphragm due to the dilated fluid-filled esophagus superimposed upon the heart.

Roentgenologic Examination. Examination of the chest showed what appeared to be considerable enlargement of the heart to the right (Fig. 1). Subsequent examination revealed that the right border of the cardiac shadow was actually due to a markedly enlarged esophagus. In the lateral view the trachea appeared to be displaced somewhat forward and there was a distinct fluid level seen in the esophagus (Fig. 2). The esophagus visualized by the barium meal was enormously dilated (Fig. 3). There was a definite obstruction at the cardiac orifice of the stomach which was smooth and regular in contour (Fig. 4). There was a large non-opaque residue in the esophagus, which was quite tortuous. *Conclusion:* Marked degree of preventriculosis, with marked dilatation and tortuosity of the thoracic esophagus. Esophagus projecting to the right side of the cardiac shadow.

Esophagoscopic Examination. There was an enormous dilatation of the entire thoracic esophagus with considerable retention of fluid.

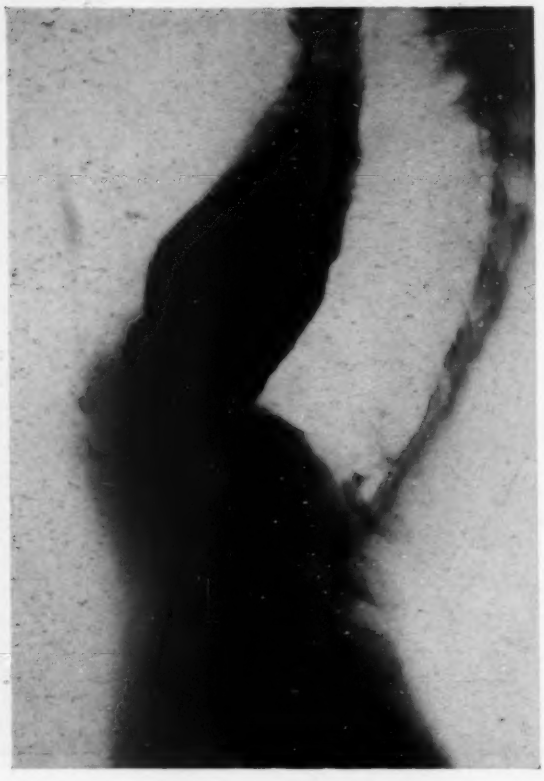

Fig. 3. Case 1. Barium-filled esophagus shows marked dilatation and tortuosity.

This dilatation extended from the cardiac orifice up to the level of the suprasternal notch. The esophagus showed tight closure at the cardiac orifice but no difficulty was encountered in passing the instrument into the stomach. The mucosa of the dilated esophagus was markedly inflamed and presented numerous superficial erosions. *Esophagoscopic diagnosis:* Preventriculosis, so-called cardiospasm.

The one outstanding condition from which achalasia of the cardia must be differentiated is carcinoma involving the lower end of the esophagus. As a rule, the differentiation is not difficult either clinically or roentgenologically. Carcinoma usually occurs after forty years of age, is more frequent in men than in women, gives a relatively short history of difficulty in swallowing which becomes progressively worse and is accompanied by definite loss in weight. In achalasia of the cardia, on the contrary, the symptoms are often intermittent in character and, as a rule, the general well being of the patient is in marked contrast to the patient with malignancy.

Roentgenologically the obstruction at the lower end of the esophagus is seldom complete. There is usually noted a continuous, much distorted stream of opaque material entering the stomach. The markedly irregular contour of the lesion is in sharp contrast to the smooth funnel-shaped appearance in achalasia of the cardia. While there is some dilatation of the esophagus proximal to the lesion this is seldom very marked. The patient takes the opaque meal a mouthful at a time and there is not the tendency to completely fill the esophagus as is so characteristic of patients with achalasia of the cardia.

While these clinical and roentgenological features readily distinguish the malignant lesion from achalasia, one occasionally encounters a case in which the two lesions

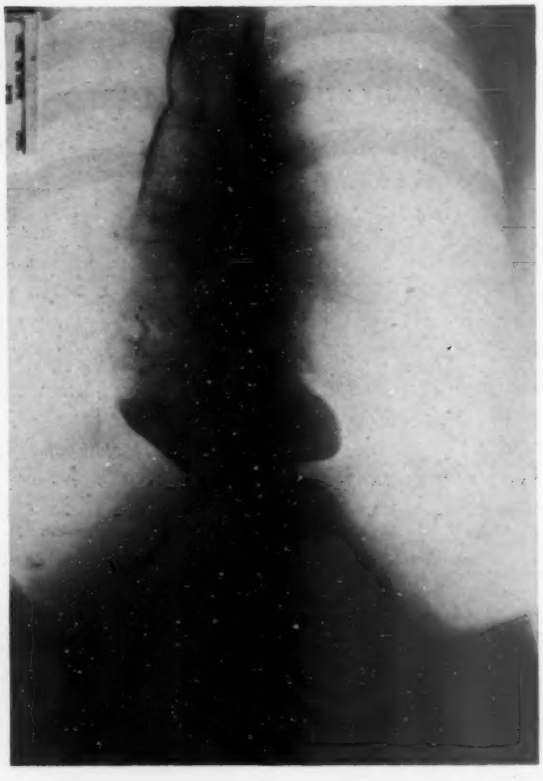

Fig. 4. Case 1. Showing the characteristically smooth and regular funnel-shaped obstruction at the lower end of the esophagus in achalasia of the cardia.

may simulate each other. The following case is an example of this kind.

Case II. M. K., female, aged twenty-six. The patient's chief complaint was discomfort beneath the ensiform cartilage for the past seven months. She began to have difficulty in eating three months ago. This was characterized by a fullness beneath the sternum and a feeling as though the food stopped before it reached the stomach. She found if she drank liquids slowly she was able to retain it, but solid foods came up just as swallowed. Getting rid of the food gave relief. For the past few weeks she had had difficulty in taking water.

Contents removed from the esophagus consisted of milky material, saliva and no blood. The blood count showed a moderate secondary anemia. The Wassermann reaction was negative.

Roentgenologic examination (Figs. 5 and 6) was reported as follows: The bony framework

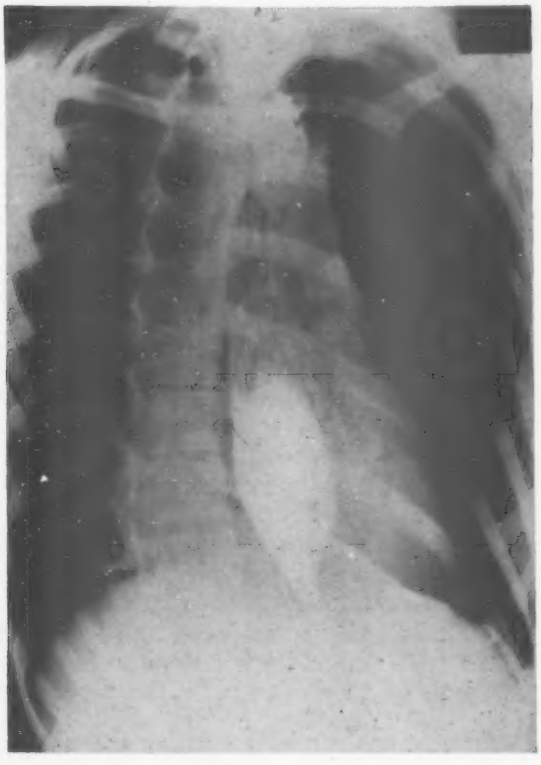

Fig. 5. Case II. This case presented the characteristic findings of achalasia of the cardia and was so diagnosed roentgenologically. Esophagoscopic examination revealed an organic stenosis of the lower end of the esophagus and at operation this was found to be due to an extensive malignancy.

Fig. 6. Case II. Lateral view of esophagus showing the appearance typical of an achalasia of the cardia in a case of cancer.

of the chest is negative. Examination of the esophagus showed a definite dilatation of the thoracic portion, with an obstruction at the cardiac orifice of the stomach. The lower end of the esophagus is smooth and regular in contour. The appearance is characteristic of a preventriculosis. We could demonstrate no organic lesion. Conclusion: Preventriculosis of a moderate degree.

Esophagoscopic Examination. The upper esophagus was normal. The thoracic esophagus showed considerable dilatation with retention of fluid. There was no barium remaining in the esophagus at the time of the examination. The dilatation extended down to the level of the diaphragm. The abdominal esophagus was closed tightly just below the level of the hiatus. The mucous membrane was very much reddened, thickened and granular in appearance. A No. 18 dilator passed through the strictured area with slight resistance. It was not thought advisable to remove tissue for biopsy. *Esophagoscopic diagnosis:* Organic stenosis of the hiatal esophagus. Neoplasm? Inflammatory? The

Fig. 7. Case III. At the first roentgenologic examination the usual findings of achalasia of the cardia were revealed. At this time the esophagus was filled with a large amount of fluid and retained food. This accounts for the mottled and irregular appearance of the esophageal shadow.

extent of the lesion and the poor condition of the patient would seem to justify a gastrostomy before further diagnostic studies are made.

At operation there was found an infiltrating cancer of the cardiac end of the stomach and esophagus. Metastasis to the liver, omentum and transverse colon. A gastrostomy was performed.

Comment. The age of the patient, twenty-six, the clinical manifestations and the roentgenological findings were characteristically those of achalasia of the cardia. The esophagoscopic examination revealed the true nature of the lesion and led to the only form of therapy that offered the patient any relief from her symptoms.

Because of the grave prognosis in cancer of the esophagus, this condition should be uppermost in the mind of the roentgenologist when called upon to examine a patient whose chief complaint is dysphagia. One is inclined to be somewhat relieved when the characteristic features of an achalasia of the cardia appear upon the fluoroscopic screen. With these findings the diagnostic problem is usually considered solved and one is not inclined to look for complicating lesions. The diagnosis of achalasia of the cardia also usually suffices for the clinician and treatment consisting, as a rule, of dilatation of the site of obstruction is proceeded with. If one bears in mind the pathology that occurs in the esophagus as the result of achalasia of the cardia the possibility of some complicating lesion would appear more probable and worth looking for. Because of the seriousness of a malignant condition the roentgenologist must make every effort to exclude this possibility when confronted by an achalasia of the cardia. The presence of such a complication can readily be missed as is illustrated by the following case.

Case III. T. M., white, female, aged forty-eight. The patient's chief complaint was pain in the abdomen. Onset of gastrointestinal upsets ten years ago at the death of her parents, with episodes of indigestion at ten and five years ago consisting of epigastric pain, nausea, vomiting and constipation. Fifty-seven pounds weight loss in ten years. An attack of "flu" five months ago with progressive downhill course characterized by dyspnea, swelling of ankles and weakness. Present digestive symptoms are of one month duration.

The blood showed a slight anemia with a slightly increased leukocyte count. Blood chemistry negative. Blood Wassermann negative. The stool was positive for occult blood.

Roentgenological examination (Fig. 7) showed the esophagus to be markedly distended from the neck to the hiatus. It was tortuous and at the dependent portion drooped. About an inch above the hiatus, the appearance on the posterior and left lateral surface suggested the presence of an ulcer. Small amounts of the barium meal were occasionally ejected into the stomach. The subdiaphragmatic portion of the esophagus was very narrow during these ejections. *Conclusion:* Esophagus—marked achalasia. We are not certain whether the point of constriction is above or below the diaphragm. We are inclined to think that it is above. The appearance suggestive of ulcer of

the esophagus near the point of constriction is probably due to mucosal folds, rather than ulcer.

Esophagoscopic Examination. The entire thoracic esophagus was enormously dilated. About 35 cm. from the upper teeth there was encountered a fungating mass the size of the end of the index finger, projecting internally from the right lateral esophageal wall. An ample specimen of tissue was removed. The lower portion of the esophagus was explored, the fungating mass producing no obstruction in the esophagus. About 44 cm. from the upper teeth there was encountered a closure of the hiatal esophagus typical in appearance of so-called cardiospasm or achalasia. The tube passed on into the stomach without undue resistance. There was no ulceration or infiltration noted as far as was examined. *Esophagoscopic diagnosis:* Achalasia, so-called cardiospasm, with closure of the lower end of the esophagus. Fungating lesion in the mid-thoracic esophagus, cancer?

A second roentgen examination (Fig. 8) was then done after complete removal of the contents from the dilated esophagus with the following result: Patient was examined after aspiration of the esophagus with the knowledge that a neoplasm had been found by esophagoscopic examination. At this time a large penetrating ulcer was found about 4 inches above the cardia. There was also a filling defect in the esophagus at this level. We should conclude from the size of the ulcer and the presence of a filling defect that the lesion at this site in the esophagus was probably a malignant neoplasm and not a benign one. *Conclusion:* Probable cancer of the esophagus associated with achalasia.

The pathological report of the biopsy was "squamous cell carcinoma, Grade 3."

This case directs our attention to two important points. The first is that the roentgenologic examination in cases of achalasia of the cardia is not complete until examination has been done after the esophagus has been evacuated of its retained contents. At the first examination, the esophagus will usually contain a variable amount of residue. While this will not interfere with arriving at the diagnosis of achalasia of the cardia, the examination should be repeated after the esophagus has been emptied in order to detect any complicating lesions.

Fig. 8. Case III. Second roentgen examination after esophagus had been evacuated of its retained contents shows a lesion just above the cardia due to carcinoma. This was missed at the first examination.

The second point is the importance of the esophagoscopic examination. As previously mentioned, no investigation of a case of achalasia of the cardia can be considered complete without direct visualization through the esophagoscope.

While one would anticipate that most of the malignant conditions associated with achalasia of the cardia would be located in the distal portion of the esophagus, this need not necessarily be the case. One must remember that secondary mucosal changes may be found in any portion of the dilated esophagus, and since the dilatation may involve the entire thoracic portion, one may expect to find malignant degeneration occurring relatively high. There is, of course, always the possibility of a carcinoma occurring quite independent of an existing achalasia. One must therefore be prepared to find a cancer in any part of the esopha-

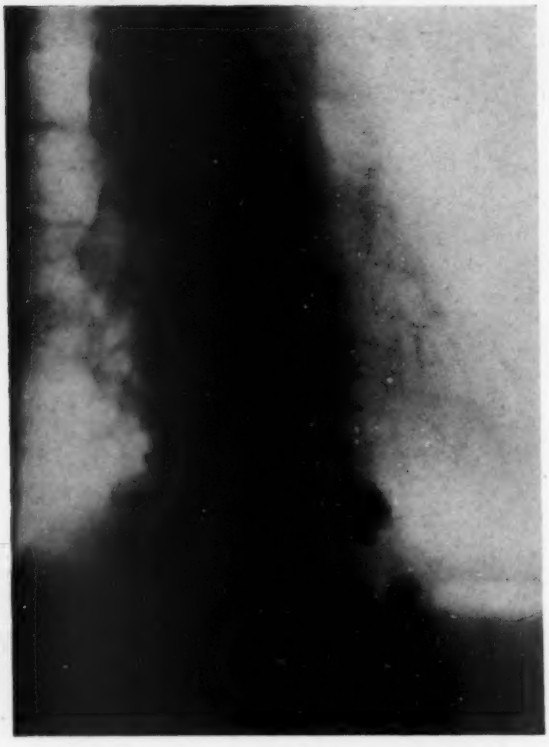

Fig. 9. Case iv. In 1926 the findings of achalasia of the cardia were confirmed by esophagoscopic examination. No complicating lesion was found at this time.

gus in association with an achalasia of the cardia, as is illustrated in the following case.

Case iv. E. S., white, male, aged forty-six. This patient was first seen in 1926 at the Jefferson Hospital at which time he was complaining of difficulty with lodgment of both liquid and solid foods, regurgitation and marked weight loss, 80 pounds. The onset was about eight years before with severe pain in the stomach and inability to retain food. One physician told him he had "autointoxication" while another treated him for gastric ulcer, with some improvement in his condition although regurgitation persisted. In 1925 he attended a hospital in another city where numerous studies were carried out and a diagnosis of cardiospasm was made.

Roentgenologic examination (Fig. 9) was carried out on November 30, 1926. There was a great dilatation of the esophagus. The obstruction was at the extreme lower end and was evidently spasmodic in character. The esophagus evidently contained a considerable amount of fluid or food at the time of the examination.

At the esophagoscopic examination on December 2, 1926, the thoracic esophagus was found enormously dilated. The mucosa was inflammatory and hard masses of food were found adherent. There was no evidence of ulceration or erosion observed. The hiatal esophagus seemed normal in appearance and a 9 mm. standard esophagoscope was passed through the hiatus into the stomach without meeting any obstruction. *Diagnosis:* Preventriculosis, so-called cardiospasm.

During the course of the next two years the patient was treated by means of hydrostatic dilatation with much improvement in his condition.

The patient was next seen at the Graduate Hospital in 1936. He complained of essentially the same symptoms as before, with the exception that he now had a cough which was thought to be due to asthma. He has continued to subsist on liquid food but his weight has definitely dropped in the past year.

Roentgenologic examination of the chest (Fig. 10) on March 6, 1936 showed a marked

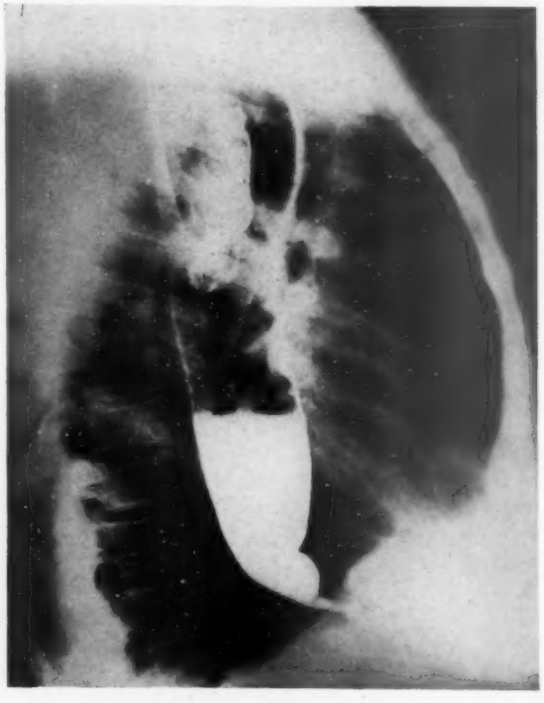

Fig. 10. Case iv. When examined in 1936 there was, in addition to the achalasia, a large carcinoma arising from the posterior wall of the upper thoracic esophagus.

degree of pulmonary emphysema. In the lateral view there was noted some anterior displacement of the trachea. Study of the esophagus revealed a hugely dilated, somewhat tortuous esophagus, the dilatation extending from the cardiac orifice of the stomach to the suprasternal notch. The appearance was typically that of a preventriculosis. Considerable amount of contents escaped from the esophagus into the stomach. In the upper portion of the thoracic esophagus along the posterior wall involving a distance of approximately 4 inches, there was a large filling defect which encroached upon the lumen of the esophagus. This was very suggestive of a malignant lesion in this region. *Conclusion:* Marked pulmonary emphysema. Advanced preventriculosis. Large filling defect involving the posterior wall of the upper thoracic esophagus probably due to malignancy.

At the esophagoscopic examination on March 7, 1936, the upper esophagus was normal. At a point just below the level of the suprasternal notch there was encountered a huge fungating mass which rose from the posterior wall of the esophagus. It presented an ulcerating surface and had the typical appearance of extensive carcinoma of the esophagus. An ample specimen of tissue was removed for biopsy. Inspection of the lower end of the esophagus showed the typical appearance of preventriculosis. A small tube was passed into the stomach. The extent of the ulceration on the posterior wall of the esophagus as outlined by the roentgen ray was confirmed. *Esophagoscopic diagnosis:* Preventriculosis. Extensive carcinoma of the upper third of the thoracic esophagus.

Pathological report of biopsy was "squamous cell carcinoma."

The last 3 cases here reported emphasize the necessity of keeping constantly in mind the possibility of carcinoma when called upon to examine a patient whose symptoms are referred to the esophagus. The relationship of cancer to achalasia of the cardia is twofold. In the first place the differential diagnosis must be made between carcinoma of the lower end of the esophagus and achalasia of the cardia, and secondly one must exclude cancer as a complication in achalasia of the cardia. There is nothing in the symptoms or physical findings to lead one to suspect an associated malignancy in a case of achalasia with the exception of the history of vomiting blood or the finding of occult blood in the stools which do not occur in uncomplicated cases of achalasia of the cardia. Therefore, a careful roentgenologic and esophagoscopic examination should constitute a part of the routine investigation of every case of achalasia of the cardia.

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SPONTANEOUS PNEUMOPERITONEUM WITHOUT DEMONSTRABLE VISCERAL PERFORATION*

By CHARLES L. HINKEL, M.D.
NEW YORK CITY

AS FAR as can be ascertained, Kelling in 1902, was the first to introduce air or gas into the peritoneal cavity for diagnostic purposes. He found that it facilitated his celioscopic observations. In 1910, Jacobaeus utilized the same principle in laparoscopy. It was not until 1912, however, that Weber of Kiew took roentgenograms following the introduction of air and oxygen into the abdomen through a trocar. Stewart and Stein in 1919 were the first to report the utilization of this method of visceral visualization in this country. Following the reports of Dandy, Carelli, and others, the literature on this subject became prolific. During the past ten years the method has been used therapeutically in some forms of intestinal and pulmonary tuberculosis.

SPONTANEOUS PNEUMOPERITONEUM

While the appearance of spontaneous pneumoperitoneum usually signifies perforation of a viscus, European writers have shown that it is not pathognomonic. Free intra-abdominal gas most often results from perforation of neoplasms or ulcers of the stomach or duodenum, the surgical significance of which is emphasized by Dehn and Pčelina, Johnson, Laurell, Copher, Friedman, and others. It is noted in the literature that pneumoperitoneum has also resulted from perforated appendices (Laurell), typhoid (Füllsack), and paratyphoid ulcerations (Dandy), carcinomata of the large bowel or rectum, traumatic rupture of the gastrointestinal tract or urinary bladder, abdominal wounds, or the presence of gas-forming bacteria in the peritoneal cavity (Bergemann). Schindler and Renshaw published a report of the accidental puncture of the stomach during gastroscopy.

Pneumoperitoneum resulting from perforation of the gastrointestinal tract, but not accompanied by peritonitis, is an unusual occurrence. It is said to be found infrequently if operation quickly follows perforation. Schnitzler reported one case of gastric ulcer and another of carcinoma of the ascending colon, both of which developed spontaneous pneumoperitoneum, and neither of which had any signs or symptoms of peritonitis. Both of these cases were attributed to perforations of the gastrointestinal tract, and both recovered without operation.

Fritz, in 1929, reported a case of almost symptomless pneumoperitoneum in a young woman who used a bulb syringe douche for vaginal cleansing. The air was thought to have entered the peritoneal cavity by way of the Fallopian tubes, as in the well known Rubin test.

The European literature contains much about the condition known as "pneumatosis cystoides intestinalis." which consists of large numbers of gas-containing bleb-like lesions in the subserosal coats of the intestines. The exact etiology is not known. The lesions usually involve the distal portion of the small intestine (Kopelowitz), but may be found almost anywhere along the gastrointestinal tract. The roentgen appearance of the intact blebs has been described by Forfota and Urban. Most writers consider the blebs to be a potential cause of pneumoperitoneum. Stenström reported a case which he believes to have been produced in this manner, although it was not pathologically verified.

PNEUMOPERITONEUM COMPLICATING PNEUMOTHORAX

Elrick, Banyai, and others, who have done extensive work with therapeutic

* From the Department of Radiology of the Presbyterian Hospital and of the College of Physicians and Surgeons, Columbia University, New York.

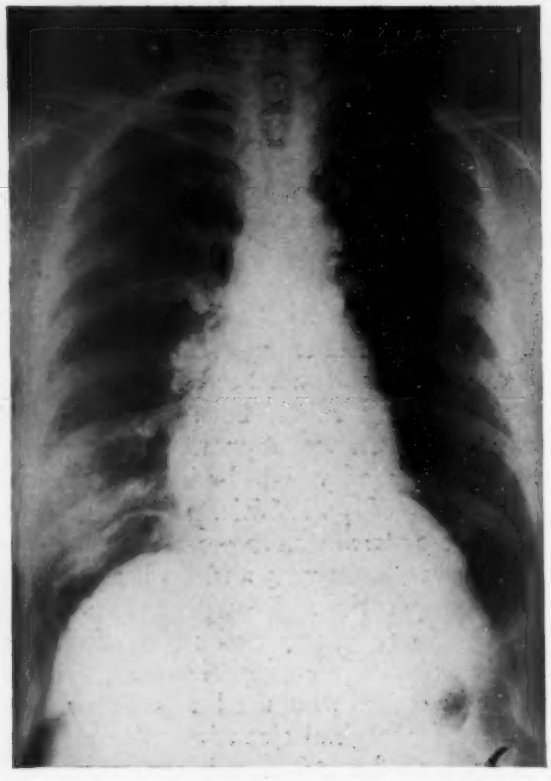

Fig. 1. Chest film three days after onset of symptoms. A large amount of free gas is present under both diaphragmatic domes. The left dome of the diaphragm is abnormally high. There is some thickening of the pleura over the left apex, and several small shadows of calcium density about the hilum.

pneumothoraces, have pointed out that infrequently pneumoperitoneum appears as a complication of this procedure. Elrick, in reviewing the English and German literature, found 11 such cases before 1929. Banyai differentiates between "direct pneumoperitoneum," in which the needle is accidentally inserted below the diaphragm and the peritoneal cavity inflated directly, and the "indirect" type. In the latter, a pneumothorax is produced, but gas is noted to appear secondarily below the diaphragm. He points out that in his 2 cases, the final intrapleural pressures were 0 and +4 mm. of water, which is higher than is usually utilized. He feels that the air entered the abdomen by way of the diaphragmatic hiati. There was no clinical evidence of peritonitis, and he subsequently maintained uneventful pneumothoraces in the same patients by keeping the intrapleural pressure subatmospheric.

SPONTANEOUS PNEUMOPERITONEUM—CAUSE UNKNOWN

Cases of spontaneous pneumoperitoneum without peritonitis and also without demonstrable visceral perforation or known exogenous origin are extremely rare. A review of the literature on this subject discloses only 2 such cases.

Moberg described the case of a woman, aged eighty-one, who developed pneumoperitoneum following prolonged vomiting. She was thought to have carcinoma of the stomach with perforation. Necropsy disclosed scirrhous carcinoma of the pylorus with marked stenosis, but there was no detectable perforation or slightest sign of peritoneal reaction. It was also discovered that there was atelectasis of the left lower lobe and terminal pneumonia at the right base. The free gas found in the abdomen was never explained, although it was suggested by Moberg that the air may have entered via the fallopian tubes while retching in a modified knee chest position.

Monod and Holiander reported the case of a Polish man, aged thirty-five, with swelling of the abdomen for eight days, who presented the clinical appearance of intestinal obstruction. At operation, gas escaped through the peritoneal incision with sufficient force to produce a loud hissing sound. The abdomen was explored through a right-sided incision, but no suggestion of peritoneal inflammation, intestinal obstruction, or other abnormality was found. The patient quickly recovered and nothing was ever disclosed to explain the presence of the tension pneumoperitoneum in this case.

The following case of unexplained spontaneous pneumoperitoneum was observed in the Presbyterian Hospital, New York City:

A Scottish housewife, aged seventy (Unit No. 157189), was admitted to the hospital on March 29, 1937, complaining of sudden, severe pain of forty-eight hours' duration in the epi-

gastrium and over the lower part of the chest anteriorly on both sides. The pain awakened her about one o'clock at night. It was sharp and stabbing in character, was aggravated by breathing, and was not relieved by soda, aspirin or a heat pad.

The patient stated that she had a dry hacking cough for twelve or fifteen years. She was thought to have had pneumonia four and one-half years previously, and for three years had noticed progressive dyspnea, substernal oppression and wheezing respirations in addition to the persistent cough.

Physical examination disclosed a slightly cyanotic and slightly dyspneic woman who did not appear dangerously ill. Temperature, pulse and respiration were normal. Blood pressure 130/68. Examination of the chest disclosed limited expansion on the left and deviation of the trachea to the right. There were absent fremitus, hyperresonance and decreased breath sounds on the left, with a few dry râles at the left base. The abdomen was distended and tender without spasm or rigidity. The remainder of the examination was negative except for evidence of generalized arteriosclerosis. Blood studies showed 69 per cent hemoglobin with 4,080,000 red blood corpuscles. The white cell count was 7,000 with a normal differential. Blood nonprotein nitrogen was 35 mg. per 100 cc., and the complete blood partition was normal. Erythrocytic sedimentation rate was 56 mm. in the first hour. Venous pressure was 65 mm. of water, the blood Wassermann negative and sputum contained no tubercle bacilli. No occult blood or other abnormalities could be found on repeated examination of the stools. Electrocardiogram revealed some evidence of myocardial damage and occasional premature contractions.

Fig. 2. Film of the abdomen taken with the patient lying on the left side and the roentgen beam directed in a horizontal plane. The normal outlines of the lateral margin of the liver and kidney are clearly seen. The shadow of the right diaphragm is not well reproduced. The position of the descending colon is indicated by a small barium residue in it.

Roentgenographic and roentgenoscopic examination disclosed a pneumoperitoneum with a large amount of free air under both diaphragmatic domes. The dome of the left diaphragm was very high, lying at the level of the fifth rib anteriorly, and was limited in excursion. The heart was shifted slightly to the right. There was evidence of old fibrotic changes and thickened pleura in both apices, but no pneumothorax was found (Figs. 1 and 2). Gastrointestinal examinations on two occasions, five and nineteen days following the onset of symptoms, disclosed no evidence of organic disease of the stomach or duodenum. No abnormality of the colon was found by repeated barium enemas. Lipiodol was instilled into the bronchial tree and it was noted that there was a peculiar pooling of the opaque material in the

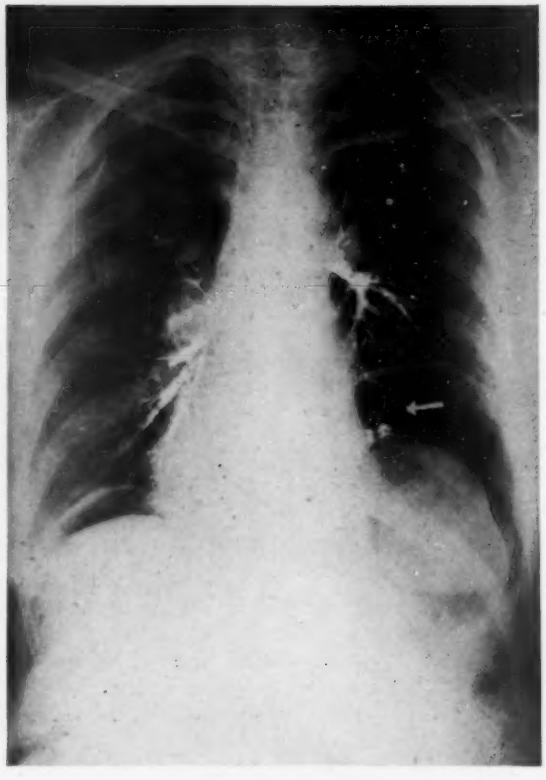

Fig. 3. Lipiodol study of the chest. The arrow points to a pocket of lipiodol which by lateral and stereoscopic films was seen to be in contact with the posterior slope of the high left diaphragm. The pneumoperitoneum, which is seen here on the eleventh day after onset of symptoms, did not completely disappear until the twenty-first day.

lowermost portion of the left lower lobe. Stereoscopic films of the chest showed the pool of lipiodol to be in direct contact with the abnormally high left diaphragm. This was interpreted as an emphysematous bulla filled with the opaque material. No lipiodol was seen below the diaphragm (Fig. 3).

The patient never became very ill. Her rectal temperature never exceeded 100° F., her white cell count never exceeded 7,000 and she improved steadily with rest in bed. Films of the chest one week after admission showed almost the same amount of pneumoperitoneum which had been present on admission. Films on the sixteenth hospital day still showed the presence of free intra-abdominal air, which did not completely disappear until the twenty-first day. She was entirely asymptomatic after the second week. On the twenty-second day she was discharged and has been followed in the out-patient department. One year later she was leading a very active life, and denied any abdominal symptoms. Roentgen studies show no change in the lung fields and no evidence of pneumoperitoneum. The erthrocyte sedimentation rate on March 20, 1938, was 24 mm. in the first hour.

DISCUSSION

The source of the gas in the peritoneal cavity of this patient remains unexplained. When questioned she stated that she had not taken a vaginal douche "in over thirty years."

No history was obtained of gastrointestinal symptoms, trauma, diagnostic air injection, or anything which might indicate the cause of this condition except a chronic cough. There was no evidence of peritonitis as judged by the clinical course, leukocyte count, temperature, or blood chemistry. The stool was guaiac negative and repeated gastrointestinal examinations and barium enemas disclosed normal findings.

The pneumoperitoneum persisted until her nineteenth day in the hospital, i.e., twenty-one days after the onset of symptoms.

Führer in 1921 studied the peritoneal absorption time of numerous gases injected into young rabbits and found this to be eighty hours per 100 cc. for pure nitrogen. Similarly accurate observations have not been carried out on humans, but experienced observers report that 1,000 to 4,000 cc. of air is absorbed in four to eight days following diagnostic pneumoperitoneum. The twenty-one day duration of the pneumoperitoneum in our case seems to indicate that the air inlet either remained open or reopened at intervals, in order to maintain the pneumoperitoneum. It is difficult to reconcile such prolonged or repeated leakage from any possible gastrointestinal source with the absence of peritonitis. In order to explain this phenomenon it must be assumed that infection did not enter with the gas.

It is undoubtedly true that some perforations of the gastrointestinal tract become "sealed off" by some means before much damage has been done. The mechanism of

this has been discussed by Warfield and others. Johnson measured a series of perforated peptic ulcers and found the average size to be 6.5 mm. while some were only 2 mm. in diameter. It is easily conceivable that a small ulcer might perforate, allow considerable gas to escape, and become sealed off again without producing much peritoneal irritation. This is what was termed "valvular pneumoperitoneum" by Singer. Even in such a case, however, it seems probable that necropsy, abdominal exploration, or satisfactory gastrointestinal examination by roentgen methods would disclose enough evidence to suggest the presence of an organic lesion if one existed.

The pool of lipiodol contiguous to the diaphragm was interpreted as possibly being an emphysematous bulla filled with the opaque material. The possibility that the pneumoperitoneum was produced and maintained by air which passed through the diaphragm was discussed. Whether this was made possible by congenital weakness in the eventrated dome, coughing, mechanical erosion, rupture of an emphysematous bulla, some local inflammatory process, or leakage around the structures in the diaphragmatic hiati cannot be determined.

The intra-abdominal pressure is nearly always negative (Wagoner) and, according to Salkin, it may reach 50 mm. of water below that of the atmosphere. The introduction of fluid or air into the peritoneal cavity does not raise the pressure sharply because of the "abdominal accommodation" described by Coombs. Therefore, if air should enter through an aperture in the diaphragm, one could not expect it to be expelled through the same route unless the intra-abdominal pressure became greater than that of the atmosphere.

The source of the gas and its mode of entry into the peritoneal cavity have not been conclusively proved, but the patient's clinical course together with the lipiodol studies suggests the possibility that there might have been at least a temporary communication between the aerated lung parenchyma and the peritoneal cavity.

SUMMARY

Spontaneous pneumoperitoneum usually results from perforation of a viscus. Two cases have previously been reported in which no cause for the free gas within the abdomen could be found.

A case of spontaneous pneumoperitoneum, which lasted twenty-one days, in a woman, aged seventy, is reported. No evidence of peritonitis or of disease of a viscus was found. Lipiodol instilled into the bronchi disclosed what appeared to be a bulla in contact with the thin, eventrated left diaphragm. As no other explanation for the pneumoperitoneum could be found, the possibility that air may have entered the abdomen by passing from this bulla through the thin diaphragm is suggested.

I wish to express my sincere appreciation to Dr. Ross Golden for his aid in interpretation of the films and assistance in the preparation of this paper.

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CARCINOMA OF THE JEJUNUM

CASE REPORT

By J. H. HARRIS, M.D., *and* J. E. GREEN, M.D.
CARLISLE, PENNSYLVANIA

THE following case is reported because of the rarity of preoperative diagnosis of carcinoma of the jejunum and because it illustrates the great importance of a careful study of the upper small intestine when vague abdominal complaints are presented.

The patient, male, single, aged twenty-seven, was referred for a gastrointestinal study in January, 1935. His symptoms consisted of discomfort in the upper abdomen with occasional vomiting. There was no pain and the history did not suggest any pathological entity. The routine roentgen study was negative.

Two years later, in January, 1937, the man was referred for a study of the colon. His symptoms had persisted and he had developed a marked anemia. A mass was palpable in the left upper quadrant. The roentgen examination of the colon was negative. On February 2, an enlarged cervical gland just above the left clavicle was removed for biopsy. The report by Dr. George Moffitt was metastatic adenocarcinoma. On February 18, an intravenous urogram showed the kidneys to be normal. The gastrointestinal tract was re-examined on February 20 and gastric peristalsis was found to be sluggish. The motility was markedly delayed, as none of the opaque mixture passed out of the stomach in thirty minutes. At the end of an hour enough barium had left the stomach to visualize the small intestine as far as the duodenojejunal junction. The duodenum was markedly dilated. At the end of three hours the barium had progressed a short distance along the jejunum and there was noted an area about 3 inches long in which all the feathery jejunal markings were obliterated and the bowel was a smooth narrow tube (Fig. 1). The roentgen diagnosis was neoplasm of the jejunum which had caused a severe grade of obstruction.

As the patient was losing weight and strength rapidly, it was felt advisable to explore the abdomen and perform a gastroenterostomy if possible. For this reason he was referred to the University Hospital, Philadelphia, where Dr. E. L. Eliason performed an abdominal section on March 5, 1937 and reported the following: "Upon opening the abdomen a mass about the size of a large orange was found in the meso of the first part of the jejunum. At a distance of 8 to 10 inches from the ligament of Treitz, there was a napkin ring constriction

FIG. 1

of the portion of the jejunum passing over the domes of this mass. The entire section of the jejunum of which the mesentery was involved by the mass was hypertrophied and edematous, due in part to obstruction and probably in part to interference with blood and lymph drainage. A short circuiting anastomosis around the point of obstruction was done by bringing a loop of jejunum distal to the mass around its lateral side to the proximal loop just beyond the ligament of Treitz."The obstructive symptoms were relieved by this operation. The patient died of inanition on September 4, 1937.

In reviewing the films of the first exami-

nation made in 1935, it was noted that a roentgenogram showing the upper small intestine was not made. At the six hour study the barium had passed the diseased portion of the bowel and hence it was overlooked. Although an hourly study of the small intestine of every case is advisable, this procedure is not feasible in many roentgen offices and departments. However, every case should have at least one film made at such an interval as to show the entire small intestinal tract. Those patients having vague complaints, in whose routine study no disease is found, should certainly have a more careful small intestinal study.

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SACCULAR ABDOMINAL AORTIC ANEURYSM*

AN ANALYSIS OF FORTY-EIGHT CASES

By M. J. HUBENY, M.D., F.A.C.R., F.A.C.P., *and* SIMON POLLACK, M.D.
CHICAGO, ILLINOIS

THE relative frequency of cases of proved saccular abdominal aneurysm in the last few years at the Cook County Hospital prompted us to study the incidence, clinical picture, and roentgenological manifestations of this interesting and variegated clinical entity.

Investigation of the hospital admissions and postmortem records revealed that since 1926 there were only 48 cases of saccular abdominal aneurysm in which the diagnosis could be considered as established beyond reasonable doubt. Twenty-eight cases, or 60 per cent, of the total were confirmed by postmortem examination; 2 of these entered in shock, died within a few minutes of admission and were diagnosed by coroner's autopsy; 3 additional cases were confirmed by surgical exploration. The remainder were selected only where the diagnosis was well established by the clinical and roentgenologic findings. We have included only the saccular types, as the dissecting aneurysms do not characteristically show diagnostic roentgen findings.

HISTORICAL

Although aneurysms as a clinical entity have long been known, the incidence of abdominal aneurysms has always been much less than those in the thoracic portions of the aorta. Ever since the first description of aneurysm of the internal vessels by Ambroïse Paré, the correlation between this condition and syphilis has been surmised. Even though Lancisi, William Hunter, and Morgagni in their classical studies of the gross anatomical features of internal aneurysm suspected a syphilitic etiology, and although they described the features of syphilitic aortitis, it was not till after Schaudinn and Hoffmann discovered the *Spirochaeta pallida* in 1905 that the etiologic importance of syphilis in aneurysm formation was proved by the demonstration of spirochetes in the involved aortic wall.

Kampmeier, in 1936, collected the cases of abdominal aneurysm from the records of the Charity Hospital of New Orleans for the past thirty years, and reviewed the literature to that date. His series included 68 cases. Thirty-eight of these were confirmed by autopsy; the others were well substantiated by clinical and roentgenologic findings. In reviewing the previous literature he referred to various collected series based both on autopsy and clinical materials, adding his 68 new cases to 313 previously reported.

Since 1935, Neely reported 5 cases in a series of 1,385 postmortem examinations at Lincoln, Nebraska.

Mills and Horton, in reviewing all the cases of aneurysm at the Mayo Clinic in the years 1935–1936 included 80 cases of abdominal aneurysm. Besides these two series, reports of 22 isolated cases were collected from the literature from the period of 1935 to 1939.

ETIOLOGICAL FACTORS

The age of the patients varied remarkably. As a rule, however, the incidence was greatest in the fourth and fifth decades. The underlying pathological change producing the weakening of the aortic wall was without doubt the determining factor for the age period of greatest incidence.

Although the general hospital admissions are only about 40 per cent colored, the incidence in our series of 71 per cent is significant and can be interpreted as a consequence of the relatively high incidence of syphilis in the colored population of this region. The increased frequency in males

* From the Department of Roentgenology, Cook County Hospital, Chicago, Illinois.

as compared to females is an old observation. Kampmeier ascribed this difference to the fact that the activity of men entails more strenuous physical exertion, which places a greater tension on the weakened aortic wall. Likewise syphilis is more common in the male.

Of the underlying pathogenic processes, by far the most common were syphilis and arteriosclerosis. It is interesting to note the variable rôle that each of these played in the different series of cases studied. In our series and in Kampmeier's, where there was a high incidence of colored patients, syphilis was the underlying cause in the majority of the cases. Seventy-five per cent of our series showed evidence of syphilis either in the serology, history, or anatomical findings. Neely, on the other hand, in reporting his 5 cases collected from a series of autopsies in Lincoln, Nebraska, could not demonstrate evidence of syphilis in any instance. All his cases were white and in the older age group, comprised of the sixth to eighth decades. Mills and Horton, in their series reported from the Mayo Clinic, found that only 8.8 per cent of the 60 cases of abdominal aneurysm showed evidence of syphilis. Only one of their patients was a negro and he had syphilis; the age of over 86 per cent of their cases was above fifty-one years. In these latter groups of cases arteriosclerosis was the predominant pathogenic factor and hence the relatively higher age group involved. Our series of white patients included 9 of 14 over sixty-two years of age and 7 of these 9 had postmortem examinations which showed evidence of arteriosclerosis and none of syphilis.

Among the rarer causative factors are mycotic emboli involving the walls of the arteries. These are usually found in the vessels of moderate caliber such as the splenic and mesenteric arteries; none of our cases were of this type. Trauma also may play a rôle as Case 1 will illustrate. This patient had no evidence of syphilis or arteriosclerosis; the blood Wassermann and Kahn reactions were persistently negative. There was a large pulsating mass in the left side of the epigastrium with audible bruit which was located at the site of the scar of an old abdominal stab wound incurred in a fight twelve years previously. Periarteritis nodosa may involve the moderate-sized abdominal arteries with resultant aneurysm formation. Wolff reports one such case showing multiple intra-abdominal aneurysms in the arteries of medium caliber. Infection from without may also involve the aortic wall and lead to aneurysm formation as illustrated by 2 cases reported by Dafoe where tuberculous aortitis and aneurysm developed secondary to adjacent caseous tuberculous mesenteric lymphadenitis. Medionecrosis cystica, a cystic degeneration of the elastic media with disruption of the elastic fibers of the aorta, is a rarer involvement and is usually associated with severe hypertension in younger individuals showing no arteriosclerosis. These processes are unusual and rare, and the only important pathogenic bases for aneurysm formation in the abdominal aorta are syphilis and arteriosclerosis. The relative importance of each of these depends on the race and age group of the patient. Syphilis is more common in the colored race and in patients up to the sixth decade; arteriosclerosis shows prevalence in white persons of the older age groups.

TABLE I

	Colored Male	Colored Female	White Male	White Female
Number of cases	26	8	12	2
Average age	45	40.4	66	38
Positive Wassermann or Kahn test	15 of 18 tested	6	2 of 8 tested	1
History or anatomical evidence of syphilis	22	7	5	0
Arteriosclerosis	0	0	7	1
Medionecrosis cystica	0	0	0	1
Traumatic	1	0	0	0
Undetermined	2	0	0	0

SYMPTOMATOLOGY

The predominant complaint in almost all these cases was pain. The usual character-

istic of this pain was progressive increase in its severity with the progress of the disease; it usually occurred in the left side of the epigastrium as the greatest number of aneurysms were found in the upper third of the abdominal aorta. As a rule, it radiated to the back in the region of the upper lumbar spine and had associated shooting radiation to the left buttock, thigh, or even the whole left extremity simulating a sciatic neuritis. The pain was often described as boring or pounding and could often be relieved, at least temporarily, by changing from a supine to a prone position. As a rule, there was no relation to the intake of food, but one patient had the typical cyclical pain of a duodenal ulcer syndrome.

Because of involvement of adjacent viscera, either by pressure or by compromising the blood supply, various symptoms of disturbance of the functions of these involved organs were present. Oftentimes these symptoms and findings obscured the presence of an aneurysm as the underlying causative factor. James reported a case of uremic coma due to compression of both renal arteries by a large abdominal aneurysm with secondary chronic interstitial nephritis. Case II in our series showed simulation of a primary kidney tumor by an aneurysm.

Gastrointestinal symptoms were often complained of as the stomach, duodenum, and colon are usually adjacent to the aneurysmal mass. Anorexia and associated weight loss were common complaints and may be a consequence of the pain. The presence of abdominal pain is in itself a finding associated most usually with inflammatory or ulcerative lesions in the upper gastrointestinal or biliary tracts. Washburn and Wilbur reported an interesting case from the Mayo Clinic which presented the clinical picture of a duodenal ulcer with pyloric obstruction and exploratory laparotomy revealed the stasis to be due to compression of the duodenum by an aneurysm.

Perforation of these aneurysms was usually posteriorly into the retroperitoneal tissues and resulted in sudden onset of excruciating pain. This is due to the hematoma dissecting up the fascial planes and disrupting the muscle fibers. Intra-abdominal, intrathoracic and intra-intestinal perforation is less common. The clinical picture of severe abdominal and lumbar pain, with associated shock and silent rigid abdomen, so simulate an acute surgical abdomen that the only 2 cases of our series who were examined in this extreme condition were both diagnosed as having perforated peptic ulcers.

The presence of other pathologic conditions may cloud the picture. Hutton and Galbraith reported a case in a taboparetic. This patient developed a paraplegia and the destructive lesion of the lumbar spine with accompanying hypertrophic changes was considered for a time to be a Charcot's spine. Case III, reported below, showed abdominal aneurysm as an incidental finding in an elderly white male, whose death was correctly attributed to a recent coronary thrombosis.

The physical findings when present were usually more reliable in arriving at a diagnosis. Of these, the presence of an expansilely pulsating abdominal mass was, as a rule, diagnostic. In a small group of the cases, the patient himself felt the mass before coming to the hospital. The expansile nature of the pulsation is peculiar and characteristic in that the palpating fingers are spread apart with each beat. This must be determined with utmost care, to rule out

TABLE II

SYMPTOMS AND FINDINGS IN FORTY-FIVE CASES

Sign	Incidence
Palpable mass	33 cases, or 71%
Expansile pulsation	30 of 32 cases with palpable mass
Bruit	17 of 20 cases where it was looked for
Thrill	8 of 15 cases where it was looked for
Pain	37 cases
a. Radiation to back	29 cases
b. Radiation to left lower extremity	7 cases

the usual type of abdominal tumors which transmit the aortic pulsation. A bruit can usually be heard over the mass and is due to the change in caliber of the vascular tube. Of the 20 cases of our series where bruit was looked for, it was present in 17. The palpation of a thrill over the mass was a more variable finding as the aneurysm wall may be thick and laminated. This finding was elicited in 8 of 15 cases where it was looked for. In those patients who showed erosion of the spine, marked localized tenderness was present over the dorsolumbar region; Murphy punch percussion over the left kidney area also showed marked tenderness in these cases. Differences in the pulsation and blood pressures in the lower extremities may contribute to the diagnosis. The findings of residual syphilitic lesions such as penile scars, syphilitic skin lesions or atrophic scarring, Argyle-Robertson pupils, perforation of the nasal septum, or absence of the deep reflexes may help in arriving at a diagnosis of the syphilitic etiology of the process.

ROENTGENOLOGIC FINDINGS

The characteristic roentgenologic findings, when present, are diagnostic and are resultant of secondary pressure defects on adjacent structures. The methods applicable in roentgen diagnosis of abdominal aneurysm include: (1) studies of the spine for erosion; (2) flat plates of the abdomen; (3) gastrointestinal studies; (4) pyelography; (5) pneumoperitoneum; (6) aortography.

By far the most common finding was erosion of the anterior portions of the vertebral bodies. Aneurysmal erosion is peculiar in that the intervertebral discs are spared giving a "scalloped" appearance to the erosion. Presumably the fibrocartilage being more resilient as compared to the vertebral bone resists the constant pressure of the aneurysm. When the descending thoracic or upper abdominal levels are involved the ribs may show erosion at their costovertebral angles. This is well demonstrated in Cases II and IV. In 10 of 23 of our series where the spine studies were carried out, diagnostic erosions were present.

Although the flat plate of the abdomen may show nothing diagnostic, there may be important findings present. A soft tissue mass may be present, possibly delineated by intestinal gas shadows; if calcification can be noted in the region of the mass (usually seen in its walls) the finding is diagnostic for aneurysm. Erosion of the spine may, if extensive enough, show up in the flat plate of the abdomen. If the mass is large enough, the iliopsoas outline is obliterated and the kidney soft tissue shadow displaced simulating the findings noted in cases of retroperitoneal tumor. Five of 8 cases in which flat plates of the abdomen were taken showed diagnostic calcifications.

The presence and localization of intra-abdominal tumors in general can often be accomplished by careful gastrointestinal studies. The roentgenoscopic examination is of most importance because not only was the mass oriented in reference to the adjacent gastrointestinal structures, but the presence of expansile pulsation could be directly observed. Pylography does not have this advantage and showed, as a rule, only extrinsic pressure displacement of the kidney (usually on the left side). Thirteen of 21 cases showed extrinsic pressure defects on the gastrointestinal or pyelographic studies.

Neither pneumoperitoneum nor aortography with thorotrast was carried out on any of these patients. Both procedures are not without certain risks which do not seem to counterbalance their possible diagnostic value.

CASE REPORTS

The following cases are reported in some detail because of the interesting clinical and pathological findings:

CASE I. J. W., a colored male, aged sixty-five, entered the Cook County Hospital June 24, 1937. He complained of persistent pain in the abdomen to the left of the umbilicus. Ever since he was stabbed in the abdomen twelve years previously, for which emergency opera-

tion was performed, he had suffered vague pains in the abdomen at the site of the scar. In the past year these pains had become more severe and constant. About that time he noted this pain radiated to the left lumbar region. In the past ten days the radiation extended with increasing severity to the left buttock and thigh. No relationship was noted between the pain and intake of food except for slight aggravation of the "misery" with large meals. The patient denied any previous venereal infection.

On physical examination the temperature was 98.6° F., pulse 80, respiration 24, and blood pressure 130/80. Both pupils reacted to light and in accommodation. The heart and lungs were essentially negative. An orange-sized mass was present in the abdomen to the left of the umbilicus with a definite expansile pulsation. The mass was not movable and no bruit was heard. The roentgenograms of the thoracolumbar spine revealed marked hypertrophic osteoarthritic changes but no evidence of erosion. The Wassermann and Kahn tests were negative on two occasions. The patient obtained some relief from analgesic medication and epidural injection and was discharged.

Case II. R. S., a colored female, aged forty-eight, first entered the Cook County Hospital on March 18, 1936, with entrance diagnosis of abdominal tumor. Her chief complaint was a persistent pain in the left lumbar region with sharp shooting radiation of this pain down her back to the left buttock. This had become progressively more severe for the past two months. Associated with the pain she had noted a mass in the left upper quadrant of her abdomen which had increased slightly in size during the past four weeks. No nocturia, frequency, hematuria or dysuria was noted. No other symptoms except progressive weakness and loss of an undetermined amount of weight were complained of.

She had had a normal menopause eight years previously. About twenty years previously she was treated for "bad blood" by arm and hip "shots."

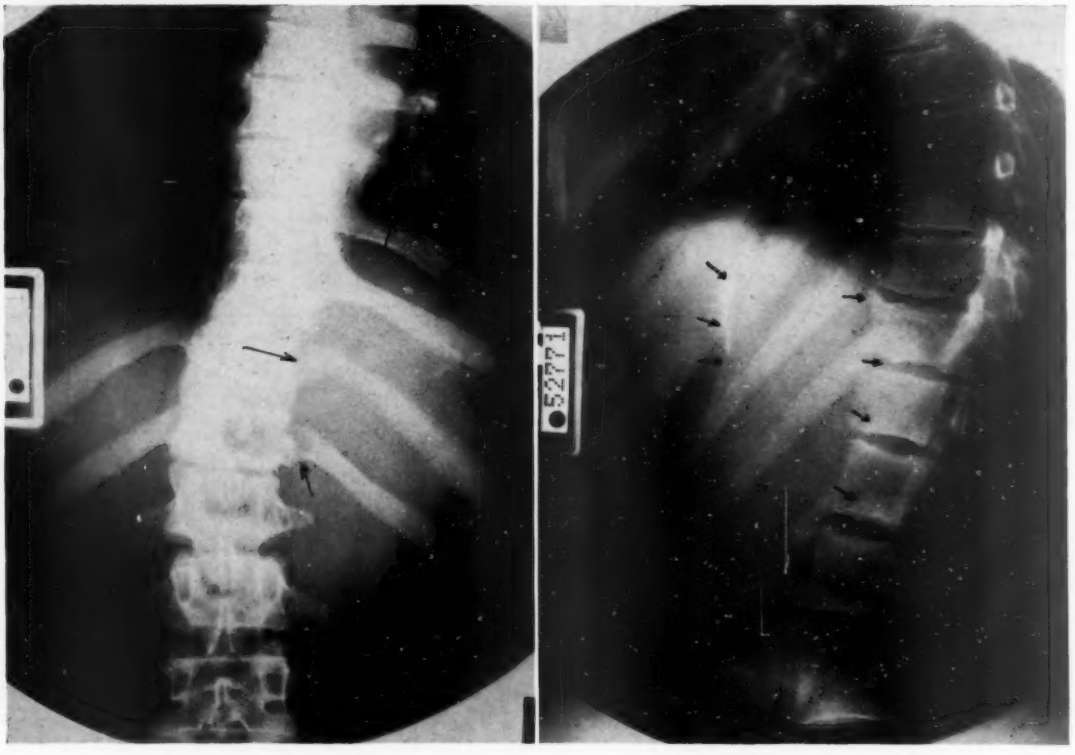
Physical examination at this time revealed a thin colored female suffering a good deal of pain. Temperature was 99.4° F., pulse 100, respiration 24 and blood pressure 110/70. The pupils were small and unequal and reacted sluggishly to light. A grapefruit-sized, movable mass was palpable in the left upper quadrant of the abdomen; no pulsation was noted at this time. Vaginal examination revealed an atrophic uterus; no connection could be made out between the pelvic organs and the abdominal mass.

The findings were considered as due to a neoplasm in the left kidney. Roentgen studies of the stomach and colon revealed no evidence of extrinsic pathology. Hemoglobin was 65 per cent, red blood count 3,050,000, white blood count 6,700. Repeated urinalysis was negative for sugar and albumin, and no microscopic or chemical evidence of urinary blood or pus was found. Repeated pyelograms showed a persistent filling defect of the upper calyces with narrowing and fuzziness of the calyces in the lower pole of the left kidney. The blood Wassermann and Kahn were both 4 plus. In spite of the absence of hematuria the genitourinary consultants considered the lesion as due to hypernephroma. After the nephrectomy incision was made and the pulsation of the mass noted, the lesion was correctly diagnosed as aneurysm of the abdominal aorta and further exploration deemed inadvisable.

The patient was discharged a few weeks later but due to the persistent severity of her pain had to be readmitted to the hosptial on October 9, 1936. She had lost a good deal of weight and was much weaker than during her previous admission. Her only relief from pain was obtained by maintaining a quiet position in bed on her left side with both legs flexed. At this time, although the blood pressure was equal in both arms, it was 140/100 in the right leg and 120/90 in the left. The heart was moderately enlarged and of aortic configuration. A faint systolic murmur could be made out and that was apical. The aortic dullness was between 6 and 7 cm. to percussion. The abdominal mass measured 10×12 cm. and had a definite expansile pulsation. A bruit was heard over it synchronous with the radial pulse. The deep reflexes were normal and no pathological reflexes could be elicited.

The films of the lumbar spine at this time (Figs. 1 and 2) revealed the characteristic erosion of the spine. The soft tissue shadow of the mass could also be made out with mottled calcification in its wall.

Because of the uncontrollable severe pain, a rhizotomy was considered for relief. On December 23, 1936, this was successfully done with resultant anesthesia on the left side to the level of D8. Ten days later on January 2, 1937, the patient died suddenly.



FIGS. 1 and 2. Case II. The large soft tissue aneurysmal mass is easily demarcated in the left upper quadrant of the abdomen. Note the erosion of the left side of the 11th and 12th dorsal vertebral bodies and the medial portions of the left 11th and 12th ribs. Diffuse calcification is present over the area of the mass. The erosion of the vertebral bodies is best noted in the lateral view.

Postmortem examination revealed the following essential anatomical findings: Huge aneurysm of the upper abdominal aorta with deep erosion of the lower thoracic and upper lumbar vertebrae and perforation through the diaphragm into the left pleural cavity; left hemothorax with compression of the left lung; brown atrophy and anemia of the myocardium; focal syphilitic aortitis of ascendens, arch and upper abdominal aorta; recent chordotomy.

CASE III. F. J., white male, aged sixty-five, entered the hospital in September 30, 1937, because of the presence of a mass in the midportion of his abdomen which had become increasingly larger in size during the previous seven weeks. In 1929 he had suffered from an illness characterized by weight loss and weakness. At that time he was examined at the Mayo Clinic and treated for hyperthyroidism. For the following six and one-half years he had been in good health. However, six to seven months preceding this hospital admission he had noticed the presence of vague abdominal pain localized to the umbilicus and not related to intake of food. In the past seven weeks he had become progressively weaker and lost about 35 pounds.

On admission, the temperature was 98 °F., pulse 88, respirations 20, and blood pressure 130/80. The pupils were round and equal and reacted to light and in accommodation. There was a large, firm, grapefruit-sized immovable mass in the epigastrium just above the umbilicus. There was a definite, expansile pulsation to the mass and a bruit was heard over it. The deep reflexes were physiological and no pathological reflexes could be elicited. There was palpable calcification of the radial arteries.

Laboratory study showed the following findings. The hemoglobin (Sahli) was 7.2 per cent, red blood cells 2,280,000, white blood cells 6,250. The blood Wassermann was negative on three occasions. Roentgen examination of the gastrointestinal tract revealed an extrinsic mass located behind and below the stomach, and above the transverse colon (Figs. 3 and 4). Expansile pulsation was noted on roentgeno-

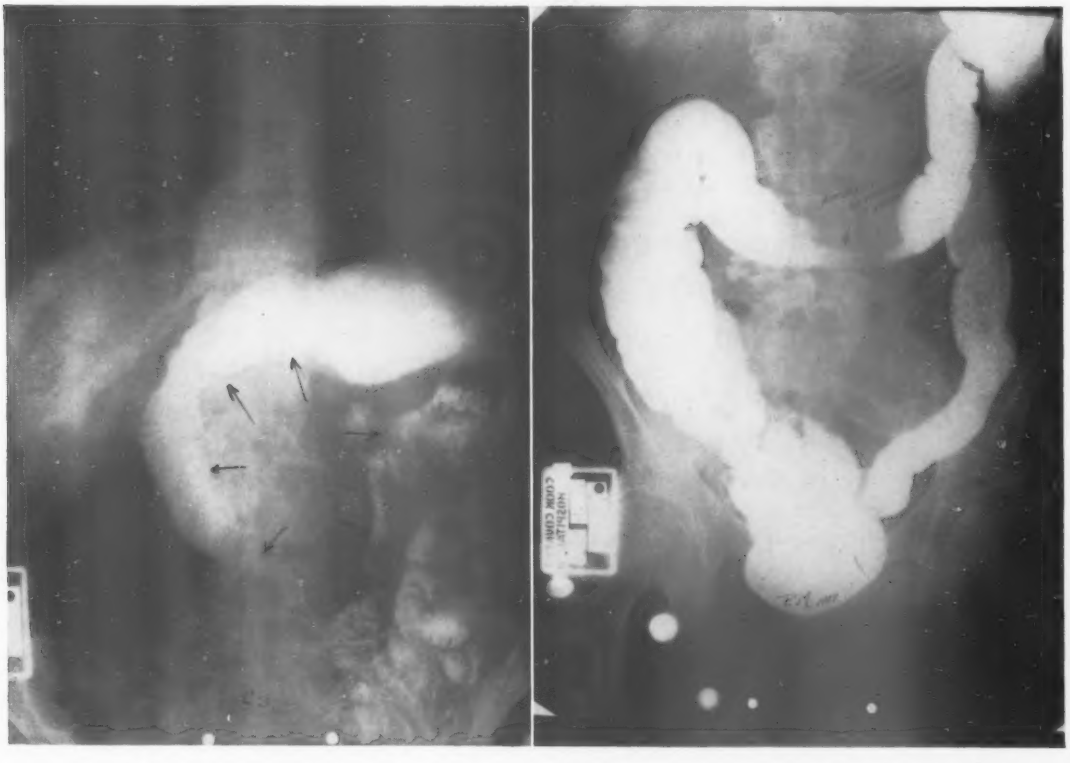
scopic examination. No erosion was noted in the lumbar spines. The electrocardiogram was interpreted as showing "auricular fibrillation with premature ventricular contractions. Evidence of severe myocardial damage. Left axis deviation. Inverted T2 and T3 seen in posterior coronary involvement."

The patient suffered from paroxysmal attacks of nocturnal dyspnea and died suddenly during one of the attacks, five weeks after admission. The clinical diagnosis was generalized arteriosclerosis with coronary sclerosis, myocardosis and arteriosclerotic abdominal aortic aneurysm.

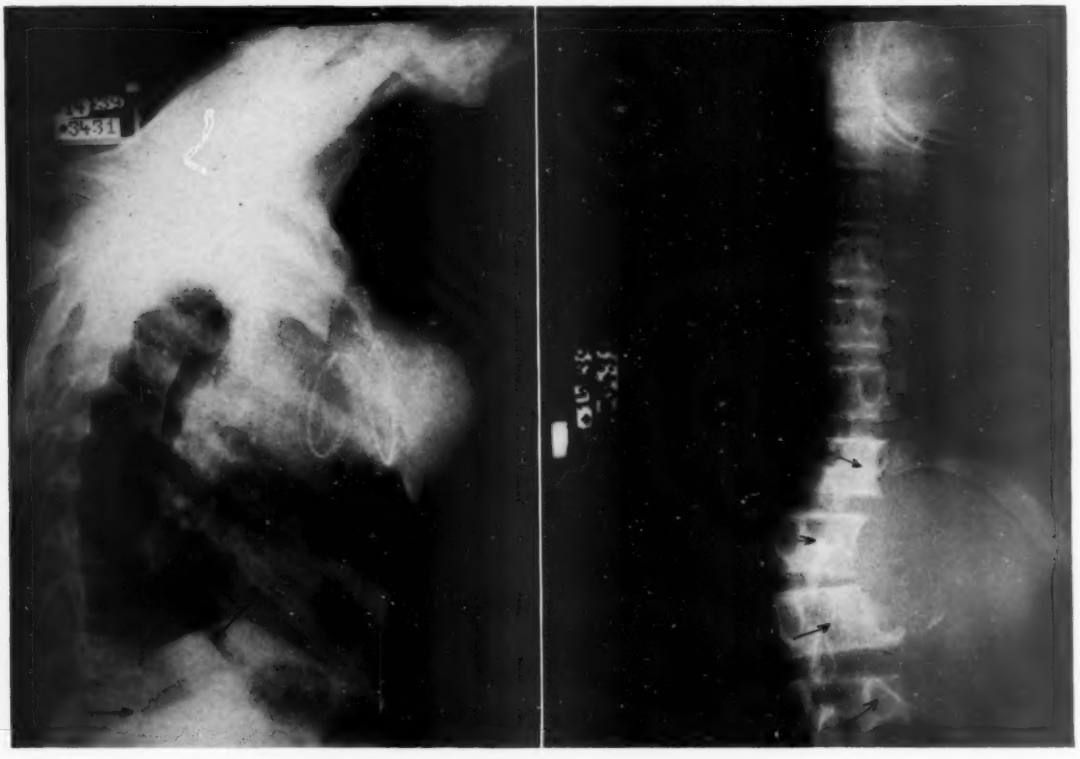
At postmortem examination the essential anatomical findings were as follows: Aneurysm of the abdominal aorta and right common iliac artery; severe atheroma of the abdominal aorta; severe sclerosis of both coronary arteries; postmyomalacic scarring of the posterior wall of the left ventricle and the interventricular septum; dilatation of both cardiac chambers; confluent bronchopneumonia of right lower pulmonary lobe; chronic cholecystitis and cholelithiasis.

Case iv. R. A., colored male, aged fifty-three, was readmitted to the hospital on March 12, 1939, as an old case of thoracic aneurysm. He gave a history of a chancre thirty-three years ago; this was treated only by cauterization. He had no difficulty with the thoracic aneurysm for almost three years after it had been wired, but about three months before this admission he noted a definite enlargement and protrusion of the chest mass and recurrence of the old steady pain. About two weeks before entrance he developed severe steady pain in the back of his left knee and leg, and the leg collapsed when he tried to bear weight on it. He was unable to get about and, because of the severe pain in both his chest and left lumbar region, had been unable to rest for the past two weeks.

On admission, the temperature was 98.6° F., pulse 80, and respirations 20. The blood pressure was 108/68 in the left arm and 98/60 in the right. The pupils were irregular in size and shape and fixed to light. There was a large protruding mass about 8×9×6 cm. in the anterior portion of the chest in the region of



Figs. 3 and 4. Case iii. The extrinsic defect pointed out by the arrows corresponds with the pulsating mass palpable and visible on roentgenoscopic examination. This is not as well demonstrated by the retrograde barium enema.



FIGS. 5 and 6. Case IV. Note the marked erosion of the left side of the dorsolumbar spine. The left 12th rib is completely destroyed. Note the wired thoracic aneurysm which has eroded through the sternum.

the upper half of the manubrium. Definite expansile pulsation, bruit, and thrill were present over this mass. There was a similar large mass in the left side of the epigastrium about 15×12 cm. in size and the expansile pulsation could be felt through to the flank. There was marked tenderness in the left flank. There was also a similar mass, fusiform in shape, about 7×10 cm. in the left iliac fossa. The left leg was enlarged and edematous; no deep reflexes could be elicited and all sensation was absent in the posterior distal two-thirds and the anterior distal half of this leg.

The laboratory studies showed 4 plus blood Wassermann. Chest roentgenogram revealed a large mass arising from the aortic arch and protruding through the eroded manubrium sterni. Numerous loops of metal were seen within this mass. There was a similar round mass in the left upper quadrant of the abdomen. The bodies of the 12th thoracic and 1st lumbar vertebrae showed characteristic erosions; the left 12th rib was almost completely destroyed (Figs. 5 and 6).

The clinical diagnosis arrived at was recurrent syphilitic aneurysm of the thoracic aorta with erosion of the sternum; aneurysm of the upper abdominal aorta with erosion of the spine and compression of the left spinal nerves; aneurysm of the left iliac artery. The patient's condition in the hospital showed steady progression in size of the masses and in his weakness and weight loss.

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NORMAL VARIATION IN THE GASTROINTESTINAL RESPONSE OF HEALTHY CHILDREN*

By ICIE G. MACY, Ph.D., LAWRENCE REYNOLDS, M.D., HELEN J. SOUDERS, M.S., *and* MARY BATES OLSON, M.S.
DETROIT, MICHIGAN

THERE is no single method completely satisfactory for determining the motility of the normal human's gastrointestinal tract, although this activity has fundamental significance in both health and disease. During growth, functional processes may be going on but there is no exact procedure for determining when the gastrointestinal pattern has become fully established; how much variation in motility exists among children within a narrow age range, when maintained under standardized conditions of environment, habit and food consumption; nor whether there exists any relation between motility and the absorption of various food components. The more recent studies in child development have made it evident that chronological age is of little value in estimating the degree of physiological maturity of an individual and that there are wide individual differences among children of a given age.

Growth is characterized by change. The healthy child may be considered as constituting "a universe in a state of continuous transformation";[11] the rate of this transformation is governed by his individual *physiological time* and is therefore the result of heredity, previous environmental factors, and other forces. While the capacity for progress in physiological time is inherent in the individual, the degree to which a child attains his optimal rate is restricted by the current factors affecting his body processes: composition and bulk of the diet; the amount of activity and physical environment; exposure to deterrent influences producing illnesses or dysfunction; habit; and, the state of mental and emotional contentment.

As a guide in timing the feeding interval, physicians[10] have used roentgen examinations to study individual variations in the gastric motility of infants and have obtained data upon the time required for complete emptying of the stomach or the rate at which the food is expelled. Some of the studies on infants have not been limited to the stomach but have been extended to include the emptying time of the segments of the intestine;[2] however, few studies have been carried out with healthy children.

Three methods of study have been used by this laboratory to obtain information upon the rate of passage of food residues through the gastrointestinal tracts of average, healthy children who were subjects of an intensive investigation of the chemistry of human growth.* Serial roentgenological examinations have been made of the progress of test meals of barium sulfate in various media through the sections of the tract, to obtain an evaluation of the response produced by different kinds of milks,[14] different types of foods (fat, protein, carbohydrate),[15] and the variation in type or speed of response at different times. The motility of the entire alimentary canal has been studied from the records of the elapsed time between ingestion and defecation of carmine markers given every fifth day for eight consecutive months to separate the fecal units for analyses, by means of which to determine the retentions of nine chemical elements.[9,19] The possible effect of carmine was studied roentgenographically.[12] The third method of study utilized the times, wet weights, and dry weights of the daily defecations during 225 consecutive days in which the same children were given a known, adequate dietary while liv-

* Data procured during longitudinal and cross-section studies of the growth and development of preadolescent children are to be published in monograph form.

* From the Research Laboratory of the Children's Fund of Michigan in cooperation with the Department of Roentgenology, Harper Hospital, and the Methodist Children's Village, Detroit.

ing in a healthful environment where habits of sleep, play, work and elimination were regular, and the frequency of defecations during twenty-six days of observation one and one-half years later.[1] These three simple methods have enabled a coördinated study to be made of the mechanics of the digestive tract in relation to other physiological factors.

By roentgenoscopic and roentgenographic observation of the serial order of events occurring in the same individual at frequent intervals following the ingestion of a single test meal into a food-empty stomach, data may be procured on the emptying time of the stomach as distinguished from the intestinal rate—the latter is quite different and far more complex to investigate than the former; on the activity of the large intestine in contrast to that of the small—the large bowel is an organ that holds back material instead of pushing it on and ejects solid material only once or twice daily while the small intestine empties liquid into the colon continuously; on the type of dispersion of a particular barium meal as well as the kind of activity it produces in the different parts of the alimentary canal; and, the amount of surface presented by the ingested material and the time of its exposure to the various digestive and absorptive processes throughout the entire digestive tract. This information permits more accurate clinical interpretations of roentgen observations in diagnosis of dysfunction in the tracts of children. Furthermore, the data contribute pertinent information to a better understanding of the physiology of the individual child as growth proceeds and aid in the interpretation of allied data on the retention and absorption of chemical elements, accumulated by metabolic balance determinations. A knowledge of the underlying physiology of the organs directly involved in the transformation of food into assimilable form and the rejection and ejection of the unused residue is fundamental in the interpretation of observations made in the clinic or laboratory.

TABLE I

PHYSICAL DATA AND EMPTYING TIMES OF STOMACHS AND JEJUNUMS OF SEVEN CHILDREN IN RESPONSE TO TEST MEALS OF BARIUM IN WATER AND IN MILK

Subject	Sex	Age	Skeletal Age*	Recumbent Length	Weight	Physical Type†	Total—174 Roentgenograms	Gastric Emptying Time		Emptying Time of Jejunum	
								Water	Milk	Water	Milk
		(mo.)	(mo.)	(cm.)	(kg.)		per child	(hr.)	(hr.)	(hr.)	(hr.)
D.P.	M	117	129	143.5	34.05	0.0226	24	1.0	3.0	1.3	3.5
W.P.	M	113	106	126.2	27.78	0.0240	25	2.0	3.0	2.5	4.0
H.H.	M	102	97	129.7	25.56	0.0230	27	2.0	1.5	3.0	2.0
F.C.	M	96	101	128.0	26.79	0.0234	24	1.8	4.0	2.3	3.0
R.S.	M	88	85	123.7	22.36	0.0228	23	2.8	2.5	3.0	3.0
B.M.	F	81	79	114.7	19.07	0.0234	27	2.5	4.5	3.0	4.5
J.H.	M	74	66	117.4	21.91	0.0238	24	1.3	3.0	1.5	3.5
Mean								1.9	3.1	2.4	3.4

* Assessed from roentgenograms (see first footnote page 396).
† Cube root of weight divided by recumbent length.

EXPERIMENTAL

The children, who served as the subjects of all the roentgenological, chemical, physiological, and clinical studies which are being correlated to appraise, in some measure, the phenomena of growth, had been under observation for several years and were known to be representative of average, healthy children; their medical histories revealed no known abnormal hereditary influences or excessive illnesses; they approximated the standards of weight for

height[2,10] (Table 1); possessed no subjective clinical symptoms, and were within the normal limits in skeletal development* and mentality.† The assessments of skeletal maturity afford a possible basis for estimating the discrepancies between the chronological and physiological ages of the subjects (Table 1).

The children were maintained in a "home unit" in the rural environment of the Methodist Children's Village where they lived as a family with their housemother and had ample opportunity for recreation with toys and playthings, indoors and out, attended school and Sunday school, and lived in a comfortable, serene, homelike atmosphere.[7,19] Their dietary, physical routine, and environment, comparable with those of the average, well child in an adequate "home" situation, were kept as constant as possible throughout all the studies. The children were familiarized with the purpose and method of each procedure, which facilitated the stimulation of regular habits and the coöperation and interest of all the subjects, thus minimizing the possibility of emotional states of fear, embarrassment, or anger, affecting the results.[20]

Each serial roentgenological study was initiated in the morning, at least one week following any previous examination. The subjects were restrained from ingesting any food or liquid (other than the test meal) between the evening meal preceding and the emptying of the test meal from the stomach. Each of the 7 subjects of this study was permitted to observe the roentgenoscopic examinations (L.R.) of the other children ingesting the water-barium meal. Each test meal consisted of 2 ounces of barium sulfate thoroughly mixed with 4 ounces of water or milk.‡ The children's interest and familiarity with the procedures made possible the exposure of the first film in each series within twelve minutes after ingestion of the test meal. Examinations were made each half hour or hour thereafter until the stomach was empty. Additional roentgenograms were procured at intervals of twenty-four, forty-eight, and sometimes seventy-two hours after ingestion of the meals (total exposures of the 7 children, 174). All of the exposures were made with the child in prone position, the tube 31 inches from the film. Exposure was $\frac{1}{5}$ sec. at 92 kv. (peak), 50 ma., with 0.5 mm. aluminum filter and Potter-Bucky diaphragm.

It has been shown by Rowe[17,18] and others[7] that a change in gastric emptying time and colonic spasm, hyperperistalsis and other irregularities in peristalsis may be due to sensitiveness of the intestinal tract to foods and for this reason, in addition to the simplicity of its preparation, it has seemed advisable to use allergen-free mixtures of barium sulfate in water in the routine examinations of patients. Because routine roentgen observations are customarily made with the water-barium test meal a knowledge of the variation in response produced by other media is essential in evaluating gastrointestinal response to the everyday mixed food dietary. The prominence of milk in the diets of infants and children and the indications that some individuals are sensitive to it make a comparative study of its effect upon the gastrointestinal motility particularly pertinent (the results of comparative studies of the response produced by other foods will be published in a following paper). To determine the permanency of the individual's particular response the milk-barium series was repeated eighteen months later.

RESULTS

The roentgenoscopic examinations with the water meal verified the fact that, in all the children, the lung fields were clear, the heart shadow normal, none of the subjects had any thymic enlargement, and shadows of the liver and spleen were normal. In each subject the size and contour of the esophagus, stomach, and duodenal bulb were within the normal range. The duodenal bulb filled immediately in all cases. In one subject (R.S.) the stomach was noted to be J-shaped and rather large for a child of his age (Fig. 1). Although the configuration of the organs was within normal limits for all subjects, variations in size, shape, and position could be seen. These observations corroborate those of Wright[22] who found in a study of 250 children, six to fifteen years old, that the position, size and shape of the stomach varies widely at all ages, showing a greater variation as puberty is approached. His observations also indicate that the sink-drain type of stomach is more common in younger children and the sharp hook type in older ones.

* Evaluation of the skeletal development of the children was made by the late T. Wingate Todd, M.D., and C. C. Francis, M.D. of Western Reserve University, Cleveland.

† Psychological examinations were made by the Children's Center, Child Guidance Division of the Children's Fund of Michigan.

‡ The milk used was pasteurized and contained 3.5 per cent fat.

The gastric and jejunal emptying times of all the children with the water-barium and milk-barium meals have been estimated from the complete series of reproductions and are presented in Table 1, together with pertinent data upon the physical measurements of the children. The average emptying time of the nutritive milk meals from the stomachs (3.1 hours) was more than 50 per cent longer than that of the inert water meal (1.9 hours); from the jejunum (3.4 hours), approximately one-third longer than that for the water (2.4 hours). The broad range of normal gastrointestinal behavior is emphasized by R.S. whose milk meal emptied from the stomach in less time than the water. However, in this case the possibility of a gastrointestinal allergy to milk cannot be discarded.[5,15,16] Although R.S. gave no apparent allergic manifestations in his previous medical history, his skeletal tissues showed irregularities in density and maturity characteristic of allergic patients,* his eosinophile count was high (an average for three different counts was 8.7 per cent whereas the average for a group of 10 children was 4.0 with a range from 1.5 to 8.7) and his skin test to milk protein was rated as one plus.

For each of the subjects the water meal required longer to empty from the jejunum (average 2.4 hours) than from the stomach (average 1.9 hours). The average emptying times of the milk meals from both stomach and jejunum (3.1 and 3.4 hours) are slightly longer, respectively, than for the water meals (1.9 and 2.4 hours). In general, the children responded characteristically to the two different types of meals: those whose stomachs emptied more rapidly with the water meal than with the milk mixture (D.P., W.P., F.C., B.M., J.H.) showed a similarly greater motility of the water meal in the jejunum; the 2 subjects (H.H., R.S.) who showed slightly greater motility of the stomach with the milk meal had as rapid motility of the jejunum with the milk as with the water meal. The variations indicated by the differences in the ranges of the subjects' response to water and milk, in both stomach and jejunum, are not significant for water or milk in either stomach or jejunum but indicate a greater variation with both meals in the jejunum.

Little or no absorption occurs from the stomach, but the gastric digested food undergoes the most profound digestive changes in the small intestine and it is here that most of the products of digestion are absorbed. Intestinal digestion begins in the duodenum about 8 to 10 cm. from the pylorus, where the secretions from the bile duct empty and combine their action with that of succus entericus, thus introducing rapid and powerful proteolytic, amylolytic and lipolytic enzymes whose activities proceed simultaneously. Digestion and absorption may continue in the large intestine, but, in addition, excretion *into* the colon may also take place.

In considering the amount of enzymatic activity to which a meal is subjected in various portions of the digestive canal, it is of interest to consider not only the rapidity and type of filling that takes place in the stomach and intestines but the absorptive processes as well. It would seem that although the length of time required for a specified meal to pass through the tract or a section of the tract will indicate a measure of its motility, which may be affected by various factors such as the size of the meal, its temperature and consistency, the same interval cannot be used in estimating the length of time the meal is exposed to the various digestive processes. Whereas the stomach is filled almost immediately upon ingestion of the meal, the emptying occurs over a much longer period of time. Although some of the meal may proceed immediately through the stomach and enter the intestine with scarcely an opportunity of being exposed to pepsin and other gastric digestive secretions, the last part of the meal may be retained until gastric digestion is quite complete before entering the intestine.

The intestine receives the contents of the stomach much more slowly than the stom-

* Evaluation of the skeletal development of the children was made by the late T. Wingate Todd, M.D., and C.C. Francis, M.D. of Western Reserve University, Cleveland.

Twelve Minutes after Ingestion
Water Meal
Milk Meal First Series
Milk Meal Second Series
Forty Minutes after Ingestion
Water Meal
Milk Meal First Series
Milk Meal Second Series
W.P.
H.H.
F.C.
R.S.
B.M.
J.H.

FIG. 1

Eighty Minutes after Ingestion

Water Meal | Milk Meal First Series | Milk Meal Second Series

Twenty-four Hours after Ingestion

Water Meal | Milk Meal First Series | Milk Meal Second Series

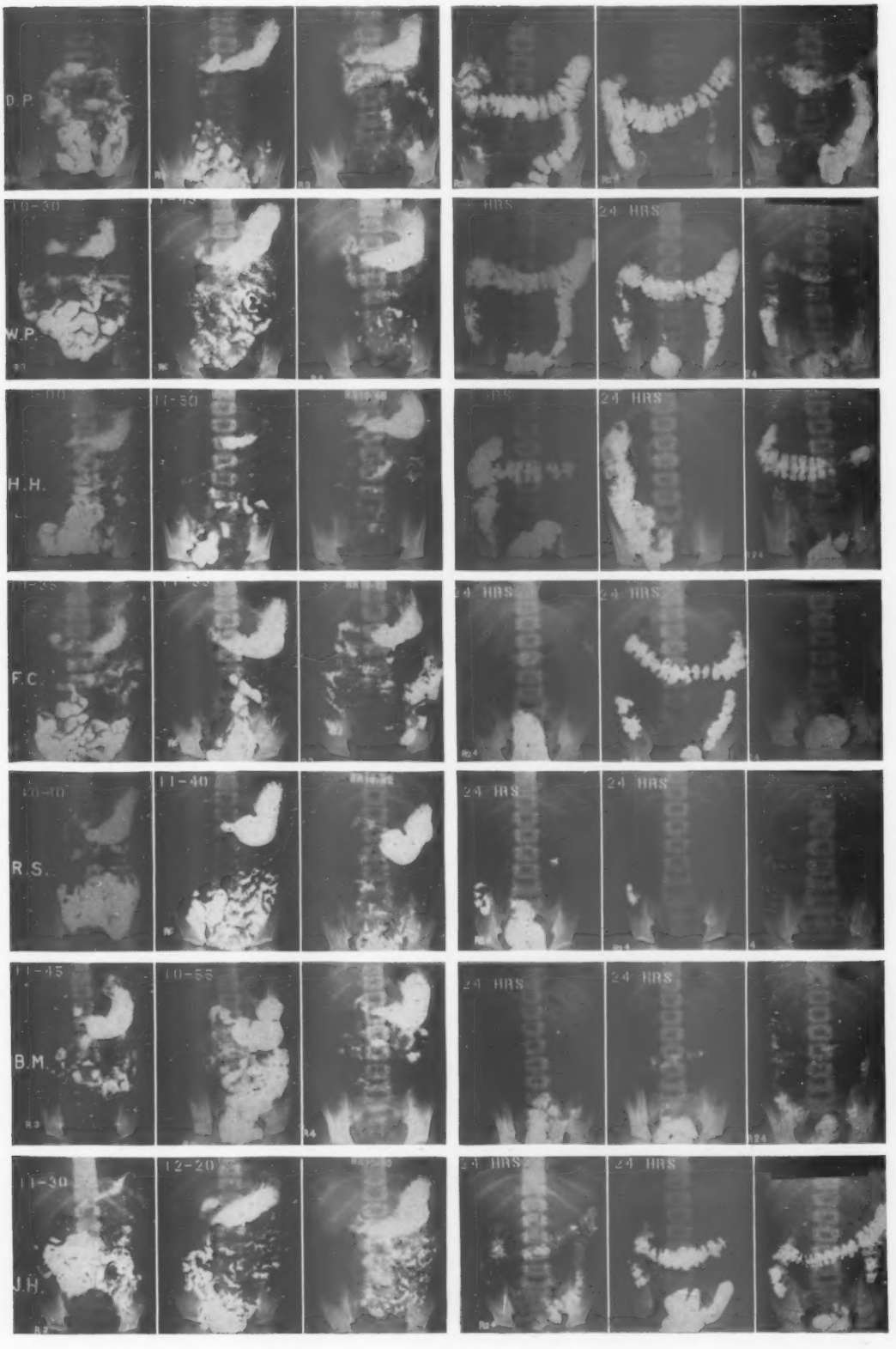

FIG. 2

ach received the original food. Each portion, as it is ejaculated from the stomach, becomes subject to intestinal action and is exposed for the entire time required to traverse that section. The time of exposure in any section of the intestines may best be represented by the interval between initial reception and emission of a portion of the test meal.

Reproductions of the roentgenograms of the 7 children with the water and milk meals at four time intervals are shown in Figures 1 and 2. The roentgenograms obtained during the second examination with the milk-barium meal are shown in the same illustrations to facilitate comparison, but have been discussed separately. The individual variations in the patterns of these average, healthy children, as well as the variations in response to water and milk as the barium media, are apparent.

In Figure 1 the rapid initiation of gastric emptying and the diffusion of the water meal into the jejunum is obvious for all the subjects except B.M. who exhibited some anxiety before taking the meal. There appears to be an intestinal as well as gastric response to the type of medium used for the barium, for the greater motility of the water meals in the stomach was accompanied by faster progress through the duodenum and jejunum. The meal consisting of water and barium excited very feeble gastric peristaltic movements, unassociated with any pylorospasm, the mixture passing quite readily through the pylorus, but not quite so completely filling the duodenal bulb as the milk meal. The water-barium meal made its egress from the stomach in a fairly orderly manner, even during the first twelve minutes following the ingestion of the meal (Fig. 1).

The small bowel pattern was quite similar in all the subjects receiving the water-barium meal (Fig. 1) with very little evidence of peristalsis, *per se*, though there seems to be a fairly rapid passage of the water-barium meal through the small intestine, and the rate of passage of this particular type of meal is fairly consistent throughout the observations of all the subjects. This may be seen in the exposures after twelve, forty, and eighty minutes.

There was an initial delay in the passage of the milk meal through the pylorus into the duodenum. Twelve minutes after ingestion of the meal, only 3 subjects (D.P., H.H., J.H.) show any of the milk-barium mixtures to have entered the small intestine, and gastric peristalsis was more vigorous than was observed with the water-barium meal. There seems to be a little more tone in the stomach filled with the milk meal, in comparison with the more or less relaxed appearance of the stomach with the water-barium meal. No inhibition of the gastric peristalsis can be observed, merely a delay in the passage of the barium out of the stomach. The small intestinal loops show considerably more peristaltic activity and churning effect than was observed with the water-barium meal, and there was a considerably longer time required for the passage of the nutritive milk-barium meal through the entire small intestine. In some subjects this delay was particularly prominent in the ileal loops. Throughout the observations the milk-barium meal produced similar patterns in the small intestines of all subjects.

The twenty-four hour exposures (Fig. 2) show that 4 subjects retained a large part of both the test meals in their colon after that interval; 1 subject's (F.C.) colon was well outlined with the milk-barium meal after twenty-four hours. The exposures made after the forty-eight hour interval show that the meals continued through the tracts and were eliminated at a rate consistent with the indications in the twenty-four hour exposures. In the group there is no apparent relationship between the length of the gastric emptying time and the motility of the entire tract. It is evident from the series of roentgenograms that the twenty-four hour exposure provides a fairly good index of the progress through the small intestine; however, it must be remembered that the twenty-four hour film was taken twenty-four hours after ingestion of the meal and that the length of time the meal was exposed to intestinal action

by the end of the twenty-four hours is the difference between the gastric emptying time and that figure.

Observation of the different patterns illustrated by the various series of pictures clearly defines the similarity in pattern between the twenty-four and forty-eight hour exposures making it obvious that any compensation or adjustment to the emptying time of the stomach must take place within the first twenty-four hours, and, therefore, largely in the small intestine where the major activities of digestion and absorption take place. The patterns of the children whose gastric emptying times were longest demonstrate a decided acceleration of passage of the meal through the small intestine, which might be an indication that more extensive digestive processes in the stomach lessen those which must be performed in the small intestine, thereby producing greater motility in this part of the tract.

Normal Variation in Gastrointestinal Response. To determine the possible range of variation which might be considered normal under clinical conditions of examination, the same milk-barium meal was observed roentgenographically in the gastrointestinal tracts of the same children one and one-half years after the first serial studies, under the same conditions of examination. While this later series of examinations was not preceded by eight months under conditions of rigid dietary and environmental control, the standardization of the subjects extended over five weeks and was more complete than would be encountered in clinical practice. Roentgenograms procured at comparative time intervals are given in Figures 1 and 2, with the two preceding series, and the estimations of gastric and jejunal emptying time are presented in Table II.

Table II

EMPTYING TIMES OF STOMACHS AND JEJUNUMS OF SEVEN CHILDREN IN RESPONSE TO TEST MEALS OF BARIUM IN MILK AT AN INTERVAL OF EIGHTEEN MONTHS

	Gastric Emptying Time		Emptying Time of Jejunum		Defecations Per Day	
Child	Examination		Examination		Examination	
	First	Second	First	Second	First*	Second†
	(hr.)	(hr.)	(hr.)	(hr.)	(hr.)	(hr.)
D.P.	3.0	3.5	3.5	4.0	1.4	1.1
W.P.	3.0	4.0	3.0	3.0	1.7	1.3
H.H.	1.5	4.0	2.0	3.5	1.7	1.2
F.C.	4.0	2.5	3.0	3.5	1.5	1.3
R.S.	2.5	4.0	3.0	3.0	2.5	2.2
B.M.	4.5	4.0	4.5	3.5	1.8	1.2
J.H.	3.0	3.5	3.5	1.0	2.0	1.4
Average	3.1	3.6	3.8	3.2	1.8	1.4

* Average during eight consecutive months of metabolic study.
† Average during the twenty-six day period within which the examinations with the milk meal were repeated.

Inspection of the figures in Table II demonstrate the great variations in response to the same test meal after eighteen months, possibly due to less complete standardization of the subjects or to changes resulting from progression of growth, with their resultant effect upon physiological maturity. The average of the gastric emptying times was increased in the second study; the average of the jejunal emptying times was decreased. The average number of defecations per day was less for each of the children during the twenty-six days within which the milk series was repeated than during the eight months of controlled study immediately preceding the first serial ex-

amination of the milk-barium meal in the gastrointestinal tract. The general average was 1.4 defecations per day as contrasted with 1.8 during the first series.

In Figures 1 and 2 the deviations in response to the repeated milk-barium meal in comparison with the earlier examinations are clear. After twelve minutes only 1 subject (F.C.) showed any of the barium to have passed out of the stomach into the small intestine. The type of peristalsis shown is practically identical, though there is a delay in its initiation. A similar orderly egress of the second milk-barium meal after gastric emptying started and the same rather slow passage of the meal through the jejunal loops may be observed. Only at the sixty minute period of observation did the jejunal and ileal loops appear fairly well outlined with the milk-barium mixture. This delay in passage of the mixture through the jejunal loops is probably explained by the slower emptying of the stomach, though in some of the subjects there seems to be considerably more segmentation of the small intestinal pattern in the later studies.

Once the barium mixture had passed through the small intestine into the large bowel, the rate of passage through the large bowel in most cases appears practically identical with the former series, except in the case of R.S., who showed an unusually rapid movement of all the meals through the large bowel compared to the other subjects. The extreme contrasts between the results of the two series accentuate the wide range of normal variation in gastrointestinal response which may be expected in roentgenological examinations of the same child at different times.

SUMMARY

Serial roentgenographic studies of the gastrointestinal response of seven children between the ages of 74 and 117 months to test meals composed of 2 ounces of barium sulfate in 4 ounces of water and in 4 ounces of milk, ingested at body temperature, produced evidence that the variation in response of different individuals, though wide, was not as great as the variation produced by the different types of media used. The observations recorded contribute pertinent information to a better understanding of the physiology and metabolism of the individual child and permit the determination of the range within which the estimation of normality may be made. They also permit a more accurate clinical interpretation of roentgen observations in diagnosis of dysfunction in the alimentary tract.

The results have greater significance because the subjects had lived together for a considerable period of time in the same highly controlled but happy environment where the food was known to contain adequate quantities of minerals,[16] vitamins B and D,[6,8] and other essential food components, and regular habits of elimination were established and known; the roentgen studies were made at the same time and at the season of the year when the alimentary tract is considered to be most active;[20] the children were experienced and trained for months in roentgenographic and metabolic technique thus enabling complete coöperation and reducing to a minimum emotional disturbances such as fear, anxiety and anger.

Milk stimulated considerably more tone and vigorous peristalsis with churning effect in the stomach and a prolonged emptying time. Furthermore, milk caused a delay in the passage of the barium through the intestinal loops, particularly in the ileal portions. There were great variations in response of the individual subjects to the milk-barium meal after an interval of eighteen months, perhaps due to physiological changes coincident with growth, or to shorter preparation, and therefore less complete standardization of the subjects.

From the 174 roentgenograms of the gastrointestinal tracts of this group of specially controlled children the mean gastric emptying time in response to the water-barium meal was 1.9 hours (range from 1.0 to 2.8); for the milk-barium meal 3.1 hours

(range from 1.5 to 4.5). The usual emptying time of the stomach is considered to vary from three to six hours, depending upon the emotional state of the individual, character of food, and other factors.[4,5,21] In all cases except one the water-barium meal entered the jejunum during the first twelve minutes after its ingestion. The emptying time of the jejunum with the milk-barium meal was 3.4 hours in contrast to 2.4 hours with the water-barium meal. There were no consistent, significant differences in the emptying of the colon as shown in the roentgenograms made twenty-four, forty-eight and seventy-two hours after the ingestion of the meals, indicating that any compensation or adjustment to the emptying time of the stomach must take place in the small intestine within the first twenty-four hours.

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THE LEAGUE OF NATIONS' WORK ON A UNIFORM PRESENTATION OF THE RESULTS OF RADIATION THERAPY IN UTERINE CANCER*

By J. HEYMAN, M.D.
STOCKHOLM, SWEDEN

AS A continuation of its previous work on a uniform presentation of the results of radiation therapy in uterine cancer, the Health Committee of the League of Nations in 1935 decided to issue annual reports on the above results. The preparation for the issue of the annual reports was entrusted to an Advisory Committee the members of which are Lieutenant Colonel A. B. Smallman of the Ministry of Health, London, Dr. A. Lacassagne, l'Institut du Radium de l'Université de Paris, and myself. Dr. Mackenzie of the League of Nations Secretariat was appointed as secretary, replaced in July, 1936, by Dr. Wasserberg.

The primary object of the proposed annual statistical reports is to provide a convenient work of reference for those who need to know what can be said statistically in regard to the results obtained by the application of radiotherapy to patients suffering from cancer of the uterus when the agreed rules for the setting-up of statistics have been observed.

The First Annual Report[1] has recently been published dealing with the results obtained in 1930 and previous years. Cancer of the cervix only has been considered.

The number of collaborators in the First report has been limited in the main to clinics and countries which were associated with the earlier work of the Radiological Sub-Commission, because the Committee has recognized that its first report must necessarily be of an experimental nature to serve as a guide to its future work.

Statements have been submitted by:

Centre des tumeurs de l'Université de Bruxelles
Belgium
(Contributed by professor J. Murdoch)
Liverpool Radium Institute, England
(Contributed by Dr. P. Malpas)
Marie Curie Hospital, London, England
(Contributed by Dr. E. Hurdon)
Radium Centre for Carcinoma of the Uterus, London County Council, England
(Contributed by Sir Comyns Berkeley)
Institut du Radium de l'Université de Paris, France
(Contributed by Dr. A. Lacassagne)
Radiumhemmet, Stockholm, Sweden
(Contributed by Dr. J. Heyman)

The number of collaborators will gradually be increased. The Committee hopes that directors of clinics and others who are interested in this subject will communicate with the chairman with a view to future participation.

The Committee also hopes that the organized international collaboration in providing statements for the annual reports will lead to a clearer conception of the problems involved and will provide the experience which is necessary for their solution.

The Committee considers it to be one of its principal tasks to provide as far as possible for a uniform presentation of the data in order that the greatest possible comparability between statements from different clinics can be secured.

Mainly because of the unlike composition of the material at different clinics a certain lack of comparability is unavoidable. This fact makes it still more urgent to defeat any unnecessary deterioration of the comparability.

In order to facilitate a uniform presentation of data the Committee has adopted certain rules which collaborators are expected to observe. The rules are included in a series of tables and relevant notes. It is my intention to report on the tables and

[1] Series of League of Nations' Publications, C. H. 1225.

* Read at the Fifth International Congress of Radiology, Chicago, Ill., Sept. 13–17, 1937.

the notes and to explain the guiding principles.

The first two paragraphs of the "Notes for Guidance of Collaborators" refer to the clinical type of cases which should be reported, namely cancer of the uterine cervix, including cancer of the stump. The Committee has considered it unsuitable to include for the present other types of uterovaginal carcinoma. Such a widening of the content of the statements must necessarily be preceded by a revision of the definitions of the different forms of uterovaginal carcinoma whereby it should particularly be considered how to differentiate between endocervical carcinoma and cancer of the body and how to register cases where such a differentiation will not be possible.

Further the statement should be limited to cases where the treatment planned was essentially radiological. The Committee has preferred not to include for instance cases where a combined surgical and radiation treatment was originally planned until sufficient experience from less complicated circumstances will be available. Those few cases which have been operated upon after failure of radiation treatment should not be counted as cured even if symptom free after operation.

Thus the following clinical types should be excluded:

1. Cancer of the corpus uteri and vagina;
2. Recurrences after radical operation;
3. Patients primarily submitted to combined operative and radiation treatment.

Cases treated previously by radiation elsewhere should also not be entered.

The third paragraph of the Notes states that "only those series of cases in which all clinical diagnoses have been microscopically confirmed can, as a rule, be accepted." The editor of the Report is empowered to invite the collaboration of institutes and individuals throughout the world but also to refuse statements which are considered not to be suitable. Paragraph 3 wants to emphasize that such statements only will be considered suitable where microscopical diagnosis is not lacking in any considerable number of cases.

Only cases microscopically diagnosed as cancer should be reported.

Not only chorio-epithelioma, sarcoma and malignant mixed tumors should be excluded but also precancerous conditions. The comparability of the statistics might be affected if the precancerous conditions were included which for the time being are very differently interpreted by the pathologists.

Paragraphs 5 and 6 stipulate:

5. The statement should relate to the total number of patients whose radiological treatment was begun during the year to which the statement refers as well as all patients examined with a view to treatment but not treated.
6. The statement should not be completed until a period of observation of at least five years from the beginning of the treatment has expired in all cases included.

These two paragraphs will be considered in the following.

Collaborators are requested to present their clinical material and the result of treatment in tabular form. On Table I should be stated:

I. The total number of patients examined with a view to treatment.

II. Of those (1) the cases *not treated* and (2) those *radiologically treated*.

It is impossible to estimate the value of statistics if the material has been incompletely reported. The results derived from such statistics cannot be reliably compared with others. With a view to comparability all cases should therefore be reported.

Patients examined at the clinic but not treated should be specified in Table II. Patients referred to the clinic by means of letter, telephone, etc., which do not allow of their being examined at the clinic are excluded. In my opinion, such cases should also be reported if we want to avoid as far as possible any unfavorable influence on the comparability of the statistics caused by voluntary selection.

The total number of cases treated should be given in Table III grouped according to stages. The number of cases in which the microscopical diagnosis is lacking should be specified for each of the four stages. Lack of microscopical diagnosis is less significant in the advanced than in the early cases. In cases cured for five years it is considered to be sufficiently significant to justify the demand for a detailed description of each individual case.

The result of treatment should be stated in Table IV estimated after a period of observation of five years. The beginning of the five year period should coincide with the beginning of the treatment which is the only period that can be satisfactorily fixed. In the first column should be entered all cases treated, in column 2 those considered as cured at the end of the five year period, in column 3 patients alive but with recurrence, in column 4 patients alive operated upon after failure of radiotherapy, in column 5 those who have died from cancer of the uterus or as a result of treatment, in column 6 patients not traced and finally in column 7 patients dead from intercurrent disease, including those having died from cancer not primarily originating from the uterus.

A presentation of data according to the above recommendations is easily accomplished provided a suitable and effective organization is available. It seems to be a reasonable demand that different clinics should observe these recommendations when publishing their results.

The last three tables have reference to the calculation of the result.

A special chapter has been devoted to the discussion of the sources of error in statistical assessment of the result of treatment. The essential ones are due to smallness of the samples for analysis and to the lack of comparability between the clinical material. I will not enter upon the procedure adopted by the Committee in order to meet the difficulties related to smallness of the samples.

The main purpose in stating therapeutic results is to allow a comparison of the results obtained at different clinics in order to estimate the value of the different methods of treatment used. Comparability is thus an essential condition which can, as mentioned above, be secured only to a certain extent.

The reason for this is that different radiotherapeutic clinics receive for treatment clinical material of unlike composition inasmuch as it contains different proportions of early or of late cases. Broadly speaking, the cause of these differences lies in the fact that, for some clinics, the material is not selected, for others a varying degree of intentional or unintentional selection takes place.

Some clinics, particularly in countries where hospital treatment is provided by the State, are responsible for the treatment of almost every patient from a defined area. There is practically no selection, the only patients not treated being those found unsuitable for treatment owing to the extent of the disease, general condition, etc. This involves nothing more than small differences which can be easily allowed for, provided all patients are included in the analysis whether treated or not.

Clinics of this type may be assumed to have non-selected and consequently comparable material. Such clinics may appropriately state the result of treatment by using the absolute cure rate, i.e., the ratio of the number of patients alive without recurrence to the total number examined, stated as a percentage. The absolute cure rate in one clinic is comparable with that of other clinics similarly circumstanced, but not with that of clinics in which selection necessarily takes place. The Committee has therefore suggested that clinics of the first mentioned type only should state the absolute cure rate.

In other clinics, a varying degree of selection may occur owing to such factors as inadequacy of facilities for treatment or of bed accommodation for all patients who present themselves, or preference of the physician who first examines the patient

for operative or radiation treatment (often depending upon the degree to which radiotherapy has developed), and probably others. In clinics of this type, the composition of the clinical material may differ considerably and the results calculated from the total number of patients would be comparable only in exceptional instances. A comparison between such clinics is justifiable only when selection similar in kind and degree takes place. To estimate that similarity in selection would, however, be difficult and is hardly possible unless detailed knowledge of the organization of the different clinics in this respect is available.

When such knowledge is available an over-all relative cure rate (calculated on the total number of patients treated) may have a certain value. It is clear, however, that over-all relative cure rates should be compared only exceptionally and with discrimination.

In order, so far as possible, to meet the difficulty of non-comparability between clinics dealing with selected material, the dividing of the cases into different groups according to the spread of the growth has been resorted to. The samples included at different clinics in one and the same stage should provide uniform material suitable for comparison. It should, however, be noticed that by "staging" the effect of selection on the comparability becomes reduced but not eliminated. Each stage contains a series of more or less advanced cases which will make a selection not only possible but —particularly in clinics with small facilities—to some extent unavoidable.

Further it should be emphasized that in staging nothing but the anatomo-clinical extent of the growth is considered whereas other factors which may affect the prognosis, and thus vitiate comparability, are entirely neglected. In this connection may be mentioned severe infection, serious disease of other organs, senescence, pregnancy and other complications which may prevent completion of treatment or ultimately cause the death of the patient before the five year period has expired. Such instances will undoubtedly occur more frequently in clinics dealing with selected material.

Another disadvantage accompanying this device for assessing comparability is that the arbitrary rules adopted for allotting cases to the various stages are interpreted by different clinicians in a different way thereby defeating still more the effect to assess the comparability of the samples. Consequently a certain lack of comparability is attached to the cure rates for different stages.

In order to reduce further the last mentioned disadvantage the Committee has proposed to supply the verbal definitions of the four stages with illustrative diagrams. The Health Organization of the League of Nations has decided to issue such an Atlas, which will probably appear in print within six months.

THE TREATMENT OF INFECTED HEMANGIOMATA*

By ERNST A. POHLE, M.D., Ph.D., F.A.C.R.
Professor of Radiology, University of Wisconsin Medical School
MADISON, WISCONSIN

RADIATION therapy has become established as the method of choice in the treatment of vascular nevi. Although there are some radiologists on the continent and perhaps a few in the United States who are using roentgen rays, radium is considered in this country to be the most valuable agent in the treatment of angiomata at present. Depending on the type of lesion we have to deal with, surface applications (plaques, screens) or interstitial radium (radon seeds, platinum filtered radium needles) are chosen and, if properly used, give good cosmetic results in approximately 75 per cent of the cases treated. This does not include the so-called nevus flammeus (port wine mark) which is not suitable for radiation therapy.

One complication which occurs occasionally in hemangioma is trauma followed by ulceration and secondary infection. A search of the literature did not reveal many references to this subject. MacKee[5] in the latest edition of his book on "X-Rays and Radium in the Treatment of Diseases of the Skin" mentions it only casually, and Andren,[1] who analyzed and reported the cases of nevi seen at the Radiumhemmet in Stockholm from 1909 to 1924 states: "If the hemangioma happens to be traumatically ulcerated when admitted, it is nowadays given immediate radiological treatment, for it has been found that the ulcerated surface clears up quickly with such treatment at the same time as the hemangioma becomes reduced in size."

In order to obtain further information on this subject questionnaires were mailed to a group of 30 well known radiologists and dermatologists throughout the country. We received 14 replies, 12 of which could be used in this survey. The following questions were asked:

1. How many cases of infected vascular nevi have you seen?
2. What is the approximate percentage of incidence in your own cases?
3. Do you recommend radium therapy of infected vascular nevi?
4. If the answer to Question No. 3 is in the affirmative, please state if you vary the dose as compared with noninfected vascular nevi.

The answers are compiled in Table 1. The majority of observers and especially those who see a large number of cases report a very small percentage of incidence of ulcerated hemangioma varying from 0 to 5 per cent. Only one physician estimated it at 10 per cent and another at 25 per cent. The majority advise radium treatment in the infected hemangioma and there is a definite tendency to use smaller doses than in the noninfected type. Several radiologists recommend the treatment of the infection by roentgen rays, followed by radium applications.

We believe the incidence of infection to be fairly low because in 152 patients with hemangiomata observed during the last decade at the Wisconsin General Hospital only 4 cases with infections were seen; 3 were angiomata and 1 was an angiofibroma.[6] Therefore, it seemed worth while to us to report our experience in their treatment.

CASE REPORTS

Case 1 (X-ray No. 3843). A female child, aged five months, was referred to the Department of Radiology on April 11, 1934, for treatment of an ulcerated vascular nevus on the inner aspect of the left thigh at the junction of the lower and middle thirds (Fig. 1). This nevus was present at birth. About a month before admission the skin in the center broke down and an ulcer developed. The entire nevus grew rather rapidly and in spite of local treatment

* From the Department of Radiology and Physical Therapy, State of Wisconsin General Hospital, Madison, Wisconsin. Read at the Twenty-fourth Annual Meeting, American Radium Society, St. Louis, Mo., May 15–16, 1939.

Table I

Answer	Question 1	Question 2	Question 3	Question 4
1	0	0	Yes	No
2	5	2.5%	Yes	Depends on size of lesion and extent of infection
3	4	2.8%	Yes	Smaller dose
4	25	25%	Yes	Surface application until ulcer is healed, then interstitial radium
5	0	0	Yes	No
6	4 or 5	Under 1%	Yes	Smaller dose
7	2	5%	After infection has subsided	—
8	1	10%	Yes	No
9	Many	10%	Roentgen rays	Smaller dose
10	12	Under 1%	No	Uncertain
11	12	?	Yes	Smaller dose
12	2 or 3	Under 1%	Yes	Roentgen rays first, then smaller dose of radium

there was no tendency to heal. The lesion was divided in two areas and a 2×2 cm. radium plaque containing 10 mg. radium element filtered through 0.1 mm. Al was applied to both areas for a period of one hour. The lesion responded well to the treatment. On May 13 a second application was given with the same technique but this time for only forty minutes. Re-examination at monthly intervals showed complete healing of the ulcer and disappearance of the hemangioma except for a few small telangiectatic areas at the edge. The child was seen last on February 24, 1939, approximately five years after the first visit; the cosmetic end-result is satisfactory (Fig. 2).

Case II (X-ray No. 5404). A female child, aged three months, was referred to the Department of Radiology on March 10, 1936, for treatment of an ulcerated vascular nevus involving part of the flexor and extensor surfaces of almost the entire left arm (Fig. 3). This lesion was present at birth and there was marked interference with motion. The family physician had treated it by means of ointments and several exposures to the quartz mercury vapor lamp. Because of the size of the lesion it was divided into several areas which were treated by means of heavily filtered radon screens

Fig. 1. Case I. Ulcerated angioma on left thigh in April, 1934.

Fig. 2. Case I. Appearance of scar in February, 1939.

Fig. 3. Case II. Ulcerated extensive angioma of left arm in March, 1936.

(equivalent to 2.0 mm. brass). Six screens were applied on March 10, 1936, at 3 cm. distance for a total dosage of 300 mc-hr. Another area was treated on September 15, 1936, with the same technique and dose, and a third area on October 14, 1936, requiring only four screens. The ulcer healed within three months after the first application and by February, 1937, most of the hemangioma had disappeared; there was full range of motion at the elbow. Residual angiomatous tissue on the dorsal left forearm was treated with seven radon screens (same technique as above) but at 1 cm. distance for a total dosage of 100 mc-hr. Re-examination in February, 1939, showed no evidence of angioma, but a soft pliable scar throughout the treated areas without interference with motion (Fig 4).

Case III (X-ray No. 4605). A male child, aged two and a half, was admitted on July 17, 1934, to the Wisconsin Orthopedic Hospital for Children. He had a large tumor in the region of the right thumb, which according to the parents had been present since birth (Fig. 5*A*). The growth measured 7 inches in circumference at the base of the thumb. There was a fluctuating erythematous swelling surrounding it and over this area there was increased local heat. Roentgen examination showed soft tissue swelling but no involvement of the bone. The fluctuating area was incised and pus was obtained. The cultures showed a *Streptococcus haemolyticus*. Tissue was removed for biopsy; the histological diagnosis was angiofibroma (Fig. 5*B*). Hot dressings were applied and on the date of discharge, October 5, 1934, the circumference of the tumor had decreased by 2 inches.

The patient was readmitted on March 16, 1935. The tumor was smaller and softer and the biopsy wound had healed (Fig. 6*A*). Radium screens were applied at a distance (five screens filtered through 0.5 mm. silver plus 1.0 mm. brass, 3 cm. distance, total dose 300 mc-hr.). Re-examination on June 30, 1935, showed no decrease in the size of the tumor and after consultation with the Department of Orthopedics it was decided to attempt excision followed by further irradiation (Fig. 6*B*). The tumor was removed and the defect skin grafted on June 2, 1936. On August 5, 1936, eight radium screens were applied to the site of the former lesion for a total dose of 300 mc-hr. The patient

Fig. 4. Case II. February, 1939. End-result after treatment.

A *B*

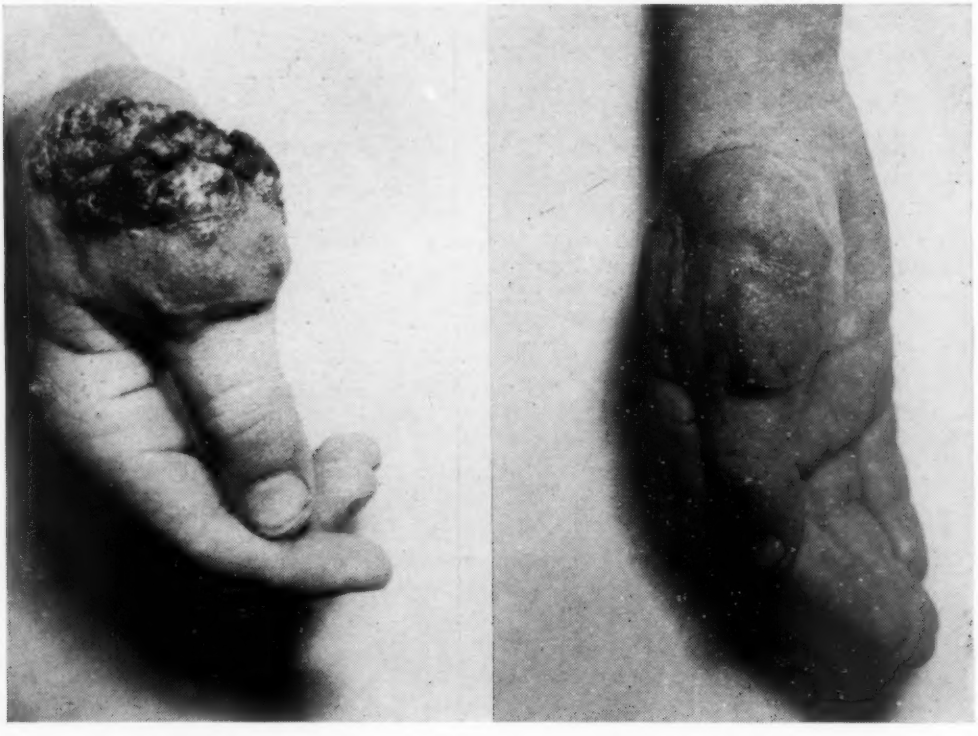
Fig. 5. Case III. *A*, angiofibroma on admission July 7, 1934; *B*, photomicrograph showing very fibrous growth with numerous blood vessels. The lumen of many of these is almost completely occluded by fibrous tissue and proliferated endothelium. (Published by permission of the *Mississippi Valley Medical Journal.*)

was seen again on February 8, 1937. The skin graft was somewhat redundant and angiomatous tissue extended into the web between the thumb and finger. The patient did not return to the hospital until June, 1938, when a massive recurrent tumor mass was present (Fig. 7*A*). Five 1 mg. radium needles of 2.5 cm. length filtered through 0.5 mm. Pt were inserted into the lesion and left in place for five days to deliver a total dosage of 600 mg-hr. The response was very gratifying and on September 30, 1938, two needles were inserted in the residual angiomatous tissue. They remained in place for seven days for a total dose of 336 mg-hr. When last seen in April, 1939, the cosmetic result seemed to be satisfactory (Fig. 7*B*). There was no evidence of recurrence and motion of the thumb was good.

A *B*

Fig. 6. Case III. *A*, appearance of growth on March 16, 1935. The biopsy wound has healed. *B*, appearance of growth on June 30, 1935, after first application of radium screens. (Published by permission of the *Mississippi Valley Medical Journal.*)

Case IV (X-ray No. 6944). A female child, aged eight weeks, was referred to the Department of Radiology on September 30, 1937, for treatment of an extensive angioma of the left forearm (Fig. 8). This lesion was present at birth and resembled then a sunburn; it grew thicker and deeper in color. In order to determine the radiosensitivity the 10 mg. radium plaque filtered through 0.1 mm. Al was applied to a selected area at a distance of 0.5 cm. for thirty minutes. When seen a month later the response had been most striking and therefore a second area was treated with the same technique. Three weeks after this application the skin broke down in both treated areas. A week later a mild hemorrhage occurred in one ulcerated area. Two weeks later further ulceration developed scattered throughout the untreated region so that this apparently had no connection with the radium treatment. The surgical consultant considered the possibility of a neoplasm and a biopsy was done. However, the microscopic examination showed only a hemangioma. The ulcers became infected and the parents decided to take the baby to another clinic for additional consultation. She was immediately hospitalized and the infected areas were treated by local applications of potassium permanganate (1–10,000) one or two times daily for two weeks, then replaced by Macey's ointment (composed of haliver oil 10 parts, cod liver oil 50 parts and 10 per cent zinc peroxide in white petrolatum 100 parts) for eleven days and then returned to the application of potassium permanganate for eleven more days during which time the infected areas continued to improve. The child was dismissed on February 18, 1938, at which time the lesion had decreased to less than the size of a silver dollar. The mother was instructed to continue Macey's ointment and to bandage the lesion. She returned with the child August 11, 1938, stating that the wound had been completely healed for four months. She had no complaint and there was no apparent limitation of motion.*



A *B*

Fig. 7. Case III. *A*, recurrence on admission, June 14, 1938; *B*, appearance of lesion on January 24, 1939, after implantation of platinum radium needles in June and in September, 1938. (Published by permission of the *Mississippi Valley Medical Journal.*)

* It was impossible to obtain a photographic record of the end-result.

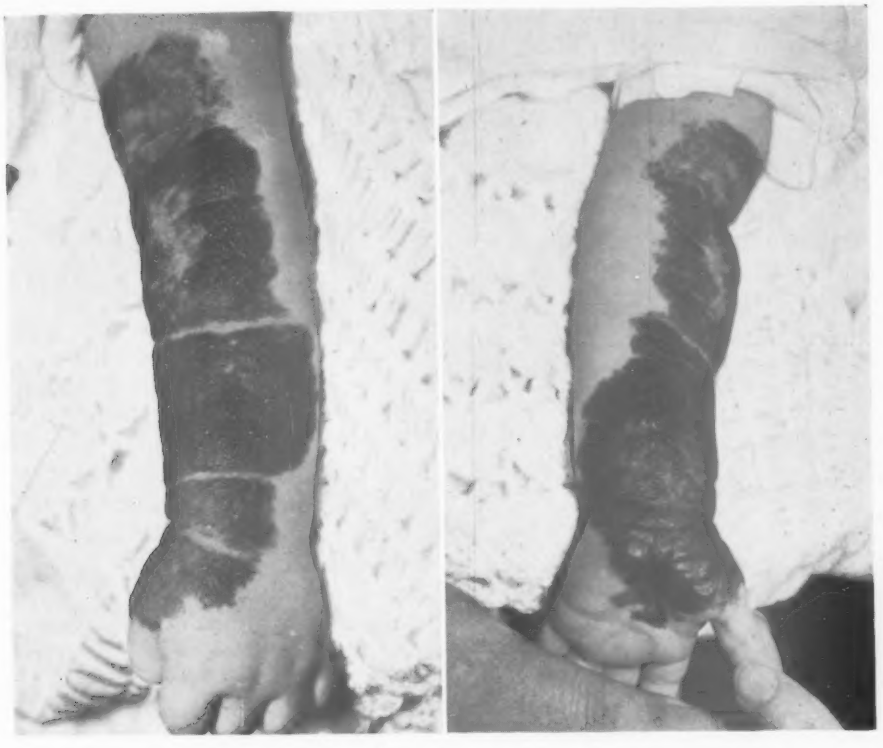


Fig. 8. Case IV. Extensive angioma on left arm in September, 1937.

COMMENT

There are a few general rules to be followed in the treatment of angiomata by radium. The superficial lesions can be taken care of successfully by means of a contact plaque containing 2.5 mg. radium element per square centimeter which is filtered through 0.1 mm. Al. For deeper lesions, heavily filtered radium screens emitting only gamma rays and applied at distances of from 1 to 3 cm. are indicated. In either case erythema reactions should be avoided and the intervals between sittings must be sufficiently long to allow the effect of a previous treatment to subside completely. It seems advisable to treat infected angiomata with smaller doses and increased intervals between exposures as compared with the noninfected type. In superficial lesions, the radium plaque may suffice as shown in our first case and satisfactory cosmetic results can be obtained. The cavernous type of angioma requires gamma rays and apparently repeated exposures over a long period. This is demonstrated in the second case which represented very extensive ulceration with involvement of parts of the flexor and extensor sides of almost the entire arm. There is no reason why roentgen rays should not be effective in treating the inflammation. Although we have no experience of our own in this respect, the procedure has been recommended by others (see Table 1).

A different approach had to be chosen in the third case. Histologically it belonged to the more radioresistant group of angiofibroma and here the ulceration followed incision of an abscess, whose etiology remained unknown. Radium surface applications were unsuccessful but implantation of radium platinum needles with low radium content brought about satisfactory regression of a massive recurrence which had developed after excision, plastic repair and external radium therapy. This method of treatment is also valuable in large cavernous angiomata as well as in lesions

which are technically difficult to handle by means of surface applicators.[2,3,4] The fourth case is interesting in view of the observation reported in the literature that cavernous angiomata occasionally regress spontaneously following trauma, ulceration or fibrosis. While the initial response to radium therapy was striking, ulceration occurred not only in the two treated test areas but diffusely in the entire involved region. Ordinary antiphlogistic treatment resulted in almost complete disappearance of the lesion without additional radium therapy.

SUMMARY

1. Radium is recognized as an efficient single therapeutic agent in the treatment of angioma (vascular nevus and cavernous angioma).

2. Infection is not a contraindication to radium therapy. However, smaller doses than those customary in the noninfected type are advocated with increased intervals between treatments.

3. Four illustrative cases of infected hemangiomata are reported. Their incidence is apparently low because in a series of 152 consecutive cases seen during a decade it corresponded to approximately 2.6 per cent.

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DISCUSSION

Dr. J. R. Driver, Cleveland, Ohio. Infected hemangiomas are not common. As a rule one is likely to see these lesions in locations similar to the ones Dr. Pohle described, for instance the inner surface of the thighs where the skin is liable to become infected by contamination with feces and urine, and I have seen these lesions a number of times on the vulva and in the intergluteal region.

Very often they are a mixed type, for example, a strawberry or capillary nevus on the surface, combined with a cavernous nevus in the base.

I agree with Dr. Pohle that irradiation helps these cases and that they heal more promptly. However, I think one should use care in the dose he employs. Smaller doses than those recommended in this paper, I believe, would be equally effective.

The healing of the infection itself has a tendency to have a beneficial effect on the hemangioma. We all know that many years ago it was the custom to vaccinate lesions of this kind in order to produce an infection, the healing of which worked out pretty well for small lesions. Naturally, it wasn't so good for the large lesions.

The treatment of hemangiomas requires individualization in many instances. It is not just a question of using radium, roentgen rays, or solid carbon dioxide. Combinations of various methods are often required.

Large capillary port wine nevi are radioresistant and respond poorly to any type of therapy. If seen within the first few weeks of life, one may use light treatments with the radium plaque or with roentgen rays. While the response may appear slight, or negligible, yet I believe such efforts may well prevent some of the late sequelae, such as the development of the deep port wine color, the late appearance of tumor-like growths, and possibly the alteration of growth of surrounding structures resulting in asymmetry.

The so-called nevus flammeus is yet another type of capillary nevus seen in the region of the center of the forehead, over the eyelids, dorsum of the nose, and nape of the neck. This type often disappears spontaneously. However, they sometimes persist. These lesions respond well to light radium treatments if given before the child is six months old.

The strawberry type of hemangioma is quite

radiosensitive if treated early. This was illustrated in the splendid result obtained by Dr. Pohle in his patient who was treated, I believe, at the age of eight weeks. Personally I would have used a smaller dose. If I understood him correctly, he used a filter of 0.1 mm. of aluminum on a half strength plaque with exposures of thirty minutes. With contact treatment this would produce a severe reaction.

Cavernous hemangiomas are generally deeply seated and may grow rapidly. They are radiosensitive and should be treated as early in life as possible, if the best results are to be obtained. Irradiation can be given by means of roentgen rays or radium. Interstitial irradiation by means of gold emanation seeds I feel is unsatisfactory. Foreign body reactions about the seeds may necessitate surgical excision later. In those cavernous hemangiomas which do not respond to irradiation, I have obtained satisfactory results from the injection of sclerosing solutions into the tumor.

Dr. Zoe A. Johnston, Pittsburgh, Pa. I have treated 6 cases of infected nevi. The locations were interesting. One involved the end of the nose in a young man; the rest were infants, one on the back in the sacral region, the other a little higher in the sacral region, and 3 were around the vulva, and inner thigh regions.

I have had the same experience as the others have had. It is better to proceed with caution when planning the treatment. Sometimes a week or two of preliminary treatment, if there is very much infection, does help to lessen the reaction following irradiation.

Dr. G. W. Grier, Pittsburgh, Pa. I saw a baby once that had a cavernous angioma on the scalp, and while the mother was trying to make up her mind whether she would have the baby treated or not, the nevus became infected, and following the healing of the infection the resultant scar obliterated the hemangioma and it didn't need treatment. Personally, I am not sure that infection of angiomas is an unmixed evil.

Dr. Pohle (closing). Regarding the dose that is used in the implantation of radium needles, this patient had in June, 1938, five 1 mg. radium needles of 2.5 cm. length filtered through 0.5 mm. Pt, inserted in the lesion and left in place for five days to deliver a total dosage of 600 mg-hr. The response was very gratifying and on September 30, 1938, two needles were inserted in the residual angiomatous tissue. They remained in place for seven days, for a total dose of 336 mg-hr.

I was asked about the disturbance of bone growth in the case where the screens are used at distance. We have never observed any, although I have no evidence in the form of roentgen studies of the bones involved. But if we can judge from growth only, there has been no untoward effect.

As regards the dosage to the child which showed ulceration, I should have stated, as in the text, that the plaque was applied at a distance of 5 mm. and at such distance we, at least, do not see any severe reactions in the skin.

Someone else asked me to mention the treatment of angiomata near the eye. We treat those, also, and if we do we make use of a set of various sized screens made of lead and plated with platinum. They are inserted over the eyeball and under the lid, and this provides complete protection. We do not get any reactions in the eyeball.

ARTHRITIS AND PARA-ARTHRITIS TREATED WITH THE ROENTGEN RAY

REPORT OF 161 CASES

By TOBIAS B. WEINBERG, M.D.
NEW YORK CITY

THE treatment of arthritis, para-arthritis and neuritis by roentgen irradiation has not received the recognition it deserves. The response of some painful joints to roentgen irradiation is frequently so dramatic that it surprises both the patient and the therapist. In recent years various forms of therapy have been tried in the treatment of arthritis as well as bursitis, neuritis and myositis. Some observers report good results with vaccine treatment; others claim cures with hyperpyrexia or radiotherm. Obviously it is difficult to evaluate the various forms of therapy because the diseases in question vary widely in their duration, their underlying causes and the pathological changes present. Therefore, very similar pathological or roentgenological changes may be associated with entirely different symptoms. Occasionally no evident symptomatology exists in spite of the advanced roentgenographic changes. For example, we frequently see spondylitic changes in the spine with associated lipping and bridging of the bodies of the vertebrae without pain, whereas similar roentgenographic findings in another case may give excruciating pains.

HISTORICAL

The treatment of arthritis with roentgen rays dates back to 1897 when Sokolow[1] first attempted it. In 1898 Stenbeck[2] reported 52 cases with 80 per cent improved. In this country Anders, Daland and Pfahler[3] in 1906 reported a great improvement following the use of roentgen treatment. Langer[4] reported good results. More recently de Lorimier[5] reported excellent results with roentgen therapy in para-arthritis.

In 1931 I started treating arthritis and other painful joint conditions with roentgen irradiation and first presented my results on December 12, 1933.* Since beginning the use of this form of treatment, over 160 cases have come under my observation and care.

FORMS OF PARA-ARTHRITIS TREATED

I shall not attempt to give a detailed history of all the cases treated but shall select a typical specimen of some of the major groups and give a short history of the case as well as the results of the treatment. In this series of cases were included arthritis, mainly of the hypertrophic variety; bursitis, the large majority of which were subdeltoid and subacromial; spondylitis involving the cervical, dorsal and lumbar spine. Some were diagnosed neuritis, whereas others were just labelled algias such as metatarsalgia, neuralgia, etc. Still others were called rheumatism.

The cases treated according to anatomical location are as follows:

1. Shoulder (a) arthritis (b) bursitis
2. Cervical spine—spondylitis
   Dorsal spine—spondylitis
   Lumbar spine—spondylitis
3. Sacroiliac arthritis
4. Knees—arthritis
5. Feet—arthritis
6. Miscellaneous: (a) torticollis (b) metatarsalgia (c) herpes zoster (d) neuralgia following extraction of teeth.

SYMPTOMS

The predominant and often the only subjective symptom was pain of variable intensity and duration. The pain was frequently excruciating and the patient in-

* Cases presented at the meeting of the East Side Clinical Society on December 12, 1933.

capacitated. Some of the associated symptoms were limitation of motion, tenderness, swelling, rarely temperature and malaise. A large percentage of these patients had positive roentgenological findings. The presence or absence of roentgenological evidence of disease, however, was not considered the criteria for instituting the treatment. Pain was always the primary indication for therapy.

HISTORY AND ETIOLOGY

The ages ranged from ten to seventy-five years; the youngest patient treated was a boy aged ten and the oldest was a man aged seventy-five. The sexes were almost evenly distributed. The duration of symptoms varied from a few days to twelve years. The most acute cases suffered only a few days; the most chronic one had symptoms over a period of twelve years. Except for the complaint in question nearly all of the patients were otherwise in good physical condition. Occasionally trauma preceded the onset of pain. In 2 patients a neuralgia of the face followed the extraction of a molar tooth. A subdeltoid bursitis was caused by a fall on the shoulder in one patient and in another a severe pain in the lumbar region was brought about by a sudden twist of the back while attempting to prevent his falling downstairs. A large percentage of the cases were either physicians or their immediate family. This fact facilitated following up many of these patients. Most of the patients had tried various other forms of therapy before being referred for roentgen treatment. Quite a number had intramuscular or intravenous medication, others had nerve block and still others had had operations for the relief of the painful condition. Most of the operations were for the removal of supposed foci of infection and included tonsillectomy, appendectomy, oöphorectomy, nasal accessory sinus drainage, dental extraction, etc. Many had tried orthopedic appliances, plaster jackets, frames, shoes and various types of splints. Most of the patients had taken therapeutic baths.

DIAGNOSIS

The diagnosis of the cases referred for therapy varied. Some were easily diagnosed such as an acutely painful shoulder with a calcified deposit in the region of the bursa. Other cases were more difficult to diagnose and included painful joints with no physical signs and no roentgen findings. Many cases were incorrectly diagnosed, a dorsal spondylitis with radiculitis being frequently thought to be a coronary disease. Cervical spondylitis with pain radiating to the shoulder and upper extremity was called arthritis of the shoulder by the physician referring the case. Some who had a lumbar spondylitis were diagnosed renal calculus and another was called metastasis. Roentgenography was helpful in clearing up the diagnosis in many cases.

PATHOLOGY

The most frequently demonstrable pathological finding was a deposit of calcium in the region of the affected joint. The deposit was either outside of the joint as seen in bursitis or visualized within the joint as in osteo-arthritis. Occasionally erosion of the joint surfaces was noted.

TREATMENT

The treatment was given directly over the involved area. The dosage was 100 to 150 r two or three times weekly and varied from three to twelve treatments. The factors were 180 kv., 4 ma., 0.5 mm. Cu plus 1.0 mm. Al, 50 cm. focal skin distance and the fields varied from 10 by 10 cm. to 15 by 20 cm., depending upon the size and the position of the involved area.

In my experience the shorter the duration of the symptoms the sooner the condition was alleviated, and the more chronic cases required a longer period of time to bring about satisfactory results. In other words, the acute cases responded to fewer roentgen treatments than the more chronic. It is essential to warn the patient that there may be a reaction following the first few treatments, associated with aggravation of symptoms, occasionally malaise and

TABLE I

Case	Name	Sex	Age	Occupation	Diagnosis	Part Affected	Duration	Treatment Began	No.	Result
1	W.N.	M	55	Physician	Bursitis	L. shoulder	Few months	12/28/31	1	C
2	J.W.	M	42	Undertaker	Calcified bursitis	R. shoulder	Few months	4/26/32	2	C
3	M.R.	M	55	Physician	Bursitis	R. shoulder		8/ 2/32	2	C
4	M.K.	M	51	Cutter	Spondylitis	Lumbosacroiliac spine	1 year	8/ 9/32	3	C
5	I.F.	M	42	Operator	Spondylitis	Lumbar spine		10/ 6/32	5	C
6	J.R.	M	60	None	Spondylitis	Lumbar spine	6 weeks	1/ 5/33	2	C
7	L.M.	F	55	Housewife	Spondylitis	Sacroiliac	3 weeks	2/ 9/33	7	C
8	E.S.	M	10	School	Torticollis	Neck	1 week	6/26/33	4	C
9	E.F.	F	42	Housewife	Spondylitis	Lumbar spine	1 year	10/ 7/33	3	C
10	L.N.	M	58	Butcher	Sacroiliac arthritis	Sacroiliac and knee	Few years	10/10/33	7	C and Rec
11	N.G.	M	30	Lawyer		L. hip	2–3 years	11/ 4/33	4	C
12	D.K.	F	51	Housewife	Spondylitis	Lumbosacroiliac spine	10 days	12/19/33	4	C
13	J.E.	M	47	Furrier	Calcified bursitis	Subdeltoid		3/ 7/34	3	C
14	R.S.	F	34	Housewife	Torticollis	Neck	Few weeks	3/14/34	3	Not imp
15	E.S.	M	10	School	Torticollis	Neck	Recurrence	3/17/34	5	C
16	R.D.	F	49	Housewife	Spondylitis	Lumbar spine	6 weeks			C
17	E.O'C.	M	40	Fireman	Calcified bursitis	L. shoulder	Few months	4/ 4/34	2	C
18	J.G.	M	50	Garageman	Arthritis	Shoulders	1 year	4/18/34	Few courses	Imp
19	V.C.	F	27	Housewife	Bursitis	L. shoulder	10 days	4/21/34	2	C
20	J.W.	M	43	Furrier	Spondylitis	Dorsal spine	Few years	4/22/34	3	Imp
21	J.C.	F	40	Housewife	Neuralgia	Face	Few days	5/ 4/34	2	C
22	L.M.	F	26	Office work	Spondylitis	Lumbar spine	Few months	5/ 9/34	1	Imp
23	J.M.	M	42	Physician	Bursitis	L. shoulder	3 weeks	5/21/34	3	C
24	L.R.	F	48	Housewife	Calcified bursitis	R. shoulder	1 week	5/28/34	4	C
25	K.F.	M	53	Housewife	Spondylitis	Lumbar spine	6 years	6/ 1/34	Course	C
26	H.F.	M	31	Business	Spondylitis	Lumbar spine	1 month	6/ 2/34	1	C
27	M.G.	M	36	Salesman	Spondylitis	Lumbar spine	5 weeks	6/ 5/34	4	C
28	G.S.	F	42	Housewife	Spondylitis	Cervico-dorso-lumbar spine	16 years	6/14/34	6	Imp
29	C.S.	M	39	Salesman	Spondylitis		3–4 days	6/23/34	1	C
30	J.G.	M	50	Business	Arthritis	Feet	Few months	7/ 9/34	5	C
31	F.S.	F	63	Housewife	Arthritis	Knees	2 months	7/14/34	3	Imp
32	J.W.	M	60	Retired	Spondylitis, arthritis	Sacroiliac, knees	1½ weeks	7/30/34	5	Imp
33	T.B.	M	57	Undertaker	Spondylitis	Lumbosacral	3 months	8/ 7/34	7	Imp
34	H.L.	M	55	Physician	Arthritis	Sacroiliac	10 years	8/15/34	3	Imp
35	A.L.	M	57	Baker	Arthritis	L. shoulder	6 weeks	8/15/34	3	C
36	M.H.	M	55	Physician	Arthritis Spondylitis	L. shoulder Cervical spine	Few weeks	8/17/34	1	Imp
37	I.L.	M	44	Dentist	Arthritis	Sacroiliac	8 days	8/27/34	Course	C
38	P.D.	M	40	Plumber	Spondylitis	Lumbar spine	Few months	9/18/34	1	C
39	A.S.	M	38	Truckman	Spondylitis	Lumbosacral	3–4 weeks	9/23/34	2	C
40	R.B.	M	40	P.O. clerk	Bursitis	R . shoulder	2 years	10/ 5/34	5	C
41	A.L.	F	50	Housewife	Spondylitis	Lumbosacral	6 months	10/ 6/34	7	Imp
42	M.K.	F	57	Housewife	Spondylitis	Cervical spine	2 weeks	10/31/34	5	C—later recurred
43	A.R.	F	44	Housewife	Arthritis	Knees and sacroiliac	3 years	11/ 8/34	9	Imp
44	R.W.	F	40	Housewife	Arthritis	R . shoulder	17 years	11/12/34	5	Imp
45	M.H.	F	59	Housewife	Arthritis	L. shoulder	5 months	11/21/34	3	Imp
46	E.K.	F	24	Maid	Metatarsalgia	Pain in the rt. first toe	Few days	11/21/34	1	C
47	J.N.	F	65	Housewife	Arthritis	L. shoulder	1 week	12/ 4/34	3	C

C = Complete relief
Rec = Recurrence
Imp = Improved

TABLE I—*Continued*

Case	Name	Sex	Age	Occupation	Diagnosis	Part Affected	Duration	Treatment Began	No.	Result
48	S.R.	M	57	Cutter	Spondylitis	Lumbar spine	Few months	12/ 8/34	5	C
49	F.D.	F	29	Housewife	Neuralgia	Following tooth extraction	Few days	12/31/34	2	C
50	A.C.	F	55	Housewife	Spondylitis	Dorsolumbar	5 years	1/ 2/35	Two courses	C
51	L.M.	M	50	Examiner	Spondylitis	Lumbar	Few years	1/20/35	1	Imp
52	S.L.	F	38	Saleslady	Spondylitis Arthritis	Cervical spine R. shoulder	6 months	2/ 5/35	Two courses	C
53	N.G.	F	55	Physician	Arthritis	Shoulders	Few years	2/ 5/35	Three courses	Imp
54	L.R.	F	47	Nurse	Arthritis	R. shoulder	3 weeks	2/ 2/35	2	C
55	M.R.	F	50	Editor	Osteo-arthritis	Shoulders	2 years	2/12/35	4	Imp
56	M.A.	F	48	Secretary	Neuralgia	Headaches	Few years	2/12/35	1	Imp—later sterilized
57	M.K.	F	43	Nurse	Spondylitis	Lumbar	15 years	2/25/35	6	Imp
58	C.T.	F	28	Stenographer	Spondylitis	Cervical	Few months	2/27/35	2	C
59	J.Z.	F	51	Housewife	Spondylitis	Lumbar	2 months	3/ 8/35	7	C
60	R.C.	F	58	Housewife	Spondylitis	Cervicodorsal	25 years	3/18/35	1	Imp?
61	M.P.	M	41	Tailor	Spondylitis	Cervical	2½ years	4/ 2/35	3	C
62	G.I.	F	65	Housewife	Spondylitis	Cervical	5 months	4/ 3/35	3	C
63	A.W.	F	50	Secretary	Spondylitis	Cervicodorsal	4 years	4/18/35	11	C
64	L.Z.	M	45	Insurance	Spondylitis	Cervical	26 days	4/22/35	5	C
65	J.S.	M	55	Business	Arthritis Spondylitis	R. shoulder Cervical	5 months	4/27/35	12	C
66	M.D.	M	61	Mechanic	Spondylitis	Lumbosacral	4 weeks	5/ 2/35	11	C
67	R.G.	F	45	Housewife	Arthritis	Hands	Few months	5/ 6/35	8	Imp
68	A.A.	F	47	Housewife	Spondylitis	Cervicodorsal	26 years	5/14/35	8	Imp
69	M.M.	F	64	Housewife	Arthritis	Sacroiliac	1 year	5/13/35	4	Imp
70	M.D.	M	41	Policeman	Spondylitis	Dorsal	4–5 years	5/13/35	4	C
71	S.S.	M	48	Salesman	Arthritis	R. shoulder	6 weeks	6/17/35	5	Imp
72	M.K.	F	39	Housewife	Calcified bursitis	L. shoulder	8 days	6/25/35	3	Imp
73	M.F.	F	38	Housewife	Spondylitis	Cervical	2 weeks	6/26/35	4	C
74	G.F.	M	65	Retired	Spondylitis	Lumbosacroiliac spine	10 months	7/ 6/35	7	Imp?
75	A.G.	M	38	Printer	Spondylitis	Dorsal spine	3 years	7/15/35	5	Imp
76	R.E.	F	75	None	Spondylitis	Lumbar	2 weeks	7/18/35	5	Imp
77	R.G.	F	44	Maid	Spondylitis	Sacroiliac	2 months	8/27/35	3	Imp
78	J.P.	M	42	Physician	Neuralgia	Headaches	Few years	9/ 3/35	3	Imp
79	B.F.	F	42	Housewife	Metatarsalgia	Right great toe	Few months	9/11/35	4	C
80	L.T.	M	55	Physician	Spondylitis	Cervicodorsal	Few weeks	9/17/35	2	C
81	R.G.	F	30	Housewife	Arthritis	Left elbow	2½ years	9/19/35	2	Imp
82	I.B.	M	27	Student	Arthritis	Sacroiliac	6 months	9/23/35	6	Imp
83	J.R.	F	40	Housewife	Spondylitis	Cervical	1 year	10/ 8/35	6	C
84	T.W.	F	42	Housewife	Spondylitis Arthritis	Cervical Shoulders	Few months	10/ 8/35	15	Imp
85	F.F.	F	50	Housewife	Arthritis	R. shoulder	1 year	10/ 8/35	3	Imp
86	F.C.	F	50	Housewife	Spondylitis	Lumbar	1 year	10/14/35	1	Imp?
87	S.S.	M	35	Business	Spondylitis	Cervical	3 months	10/31/35	6	Imp
88	E.S.	M	60	Business	Spondylitis Arthritis	Lumbar Sacroiliac	3 years	11/21/35	5	C
89	M.B.	M	55	Physician	Arthritis	Elbows	Few months	11/ 2/35	2	Imp
90	S.K.	F	48	Housewife	Spondylitis	Cervical	1 month	11/26/35	2	Imp
91	M.R.	F	35	Nurse	Spondylitis	Lumbar	3–4 months	12/ 9/35	2	Imp?
92	J.M.	M	50	Dentist	Calcified bursitis	L. shoulder	Few months	12/10/35	4	Imp
93	M.L.	M	55	Physician	Arthritis	L. shoulder	Few months	12/19/35	4	C
94	T.Z.	F	55	Butcher	Spondylitis	Cervical	Few months	12/27/35	7	C
95	E.S.	M	42	Physician	Spondylitis	Cervical	Few weeks	12/23/35	3	Imp
96	I.G.	F	44	Housewife	Spondylitis	Cervical	Few months	1/28/36	6	C
97	E.S.	M	32	Physician	Spondylitis	Cervical	Few weeks	2/ 1/36	4	C
98	A.B.	F	75	Housewife	Spondylitis	Cervical	Few weeks	2/25/36	2	C
99	A.G.	M	60	Physician	Shingles	Chest	Few weeks	3/16/36	2	C
100	R.G.	F	40	Maid	Spondylitis	Lumbar	Few months	3/30/36	7	C

TABLE I—*Continued*

Case	Name	Sex	Age	Occupation	Diagnosis	Part Affected	Duration	Treatment Began	No.	Result
101	I.M.	F	35	Housewife	Spondylitis	Cervical	1 year	4/ 8/36	5	C
102	H.R.	M	40	Lawyer	Spondylitis	Cervical	Few months	4/26/36	4	Imp
103	S.G.	M	40	Physician	Spondylitis	Cervical	3–4 months	5/ 1/36	2	C
104	J.A.	M	50	Physician	Herpes zoster	Chest and L. shoulder	Few months	6/16/36	6	C
105	M.R.	M	40	Dentist	Spondylitis	Cervical	2 weeks	8/18/36	2	C
106	A.B.	F	55	Housewife	Spondylitis	Lumbar	2 months	12/ 5/36	6	Imp
107	M.H.	F	35	Housewife	Arthritis	R. sacroiliac	1 year	12/19/36	11	Imp
108	A.L.	F	55	Housewife	Arthritis	R. shoulder	Few months	12/20/36	6	Imp
109	E.R.	F	55	Housewife	Arthritis	All joints	Few years	1/ 6/37	21	Imp?
110	J.F.	F	48	Housewife	Arthritis Spondylitis	Shoulders Cervical	1 year	1/ 7/37	13	C
111	Y.G.	F	38	Housewife	Arthritis	Sacroiliac	Few weeks	2/21/37	3	C
112	A.S.	M	55	Clerk	Arthritis	Right hip	Few weeks	2/13/37	12	C
113	S.G.	M	55	Retired	Arthritis Spondylitis	L. shoulder Cervical	3 months	4/ 2/37	10	Imp?
114	B.K.	F	21	Housewife	Calcified bursitis	Subacromial	Few weeks	4/ 4/37	2	C
115	I.H.	M	32	Dentist	Calcified bursitis	Subacromial	Few weeks	4/24/37	4	C
116	R.M.	F	36	Housewife	Spondylitis	Cervicodorsal	1 year	5/ 4/37	10	C
117	J.B.	M	35	Mechanic	Arthritis	L. shoulder	1 year	5/ 6/37	10	Imp
118	R.S.	F	60	Housewife	Spondylitis	Lumbar	Few years	5/ 7/37	14	Imp
119	S.K.	M	65	Retired	Arthritis	L. shoulder	3 months	5/10/37	3	Imp
120	J.R.	M	45	Garageman	Spondylitis	Lumbar	2 months	5/11/37	4	C
121	W.G.	M	40	Salesman	Spondylitis	Lumbosacral	Few months	5/11/37	6	Imp?
122	S.G.	M	50	Business	Spondylitis	Lumbar	1 year	5/28/37	15	Imp
123	L.G.	F	35	Housewife	Arthritis	Right elbow	Few weeks	6/ 8/37	4	C
124	A.G.	M	60	Porter	Spondylitis	Lumbosacral	10 years	6/ 8/37	8	C
125	A.D.	M	35	Printer	Spondylitis	Lumbosacroiliac	Few months	6/17/37	6	C
126	S.W.	M	38	Salesman	Spondylitis	Cervicodorsal	3 years	6/17/37	7	Imp
127	L.H.	F	35	Saleslady	Spondylitis	Cervicodorsal	2½ weeks	6/24/37	7	C
128	L.N.	M	60	Butcher	Arthritis	Left sacroiliac and knee	Few years	7/ 6/37	Three courses	Imp
129	M.B.	M	45	Physician	Calcified bursitis	Subdeltoid	Few days	7/ 7/37	7	C
130	M.M.	M	25	Salesman	Bursitis	R. shoulder	5 months	7/16/37	4	C
131	R.G.	F	34	Housewife	Spondylitis	Cervical	4 months	7/28/37	6	C
132	B.W.	M	60	Physician	Spondylitis	Lumbar	Few years	8/10/37	8	Imp
133	M.G.	M	40	Executive	Spondylitis	Cervical	Few months	8/10/37	8	Imp
134	M.S.	F	35	Saleslady	Spondylitis Arthritis	Cervicodorsal R. sacroiliac	2 months	8/19/37	10	Imp
135	S.N.	F	70	Housewife	Spondylitis	Cervical	4 months	8/28/37	9	Imp
136	P.G.	F	65	Housewife	Arthritis	Knee and ankle	Few months	9/ 3/37	3	Imp
137	S.M.	M	35	Truckman	Arthritis	Sacroiliac	4 months	9/ 7/37	10	Imp
138	M.K.	F	60	Housewife	Spondylitis	Cervical	4 weeks	9/18/37	8	Imp
139	I.R.	M	42	Physician	Bursitis	Shoulder	Few weeks	3/10/37	4	Imp
140	B.G.	F	32	Housewife	Metatarsalgia	Great toe	1 year	4/ 7/34	3	C
141	J.W.	M	41	Butcher	Spondylitis Arthritis	Lumbar Sacroiliac	8 years	12/14/34	9	C—Rec
	J.W.	M	41	Butcher	Spondylitis Arthritis	Lumbar Sacroiliac	1 week	6/21/35	7	C
142	T.K.	M	39	Laborer	Spondylitis	Lumbosacral	4 weeks	10/26/37	4	Imp?
143	M.K.	F	56	Housewife	Spondylitis	Cervical	5 weeks	1/10/38	5	Imp
144	M.T.	M	36	Grocer	Arthritis	Sacroiliac	9 months	2/17/38	14	Imp
145	J.K.	M	62	Watchman	Arthritis	Sacroiliac	2 months	2/24/38	4	C
146	J.S.	F	52	Housewife	Bursitis	Shoulder	5 days	2/23/38	6	C
147	A.L.	F	56	Housewife	Arthritis	Sacroiliac	2 years	3/ 8/38	6	C
148	E.B.	F	60	Housewife	Osteo-arthritis	Knee	9 months	3/15/38	8	Imp
149	E.G.	F	40	Housewife	Arthritis	Shoulder	2 years	3/16/38	10	Imp
150	L.S.	M	29	Hat-blocker	Spondylitis	Dorsal	2 years	3/18/38	2	Imp
151	B.A.	M	50	Storekeeper	Spondylitis	Lumbar	5 weeks	4/ 1/38	8	Imp
152	J.G.	M	49	Dentist	Bursitis	Shoulder	4 weeks	4/16/38	4	C
153	N.S.	M	48	Presser	Bursitis	Calcified sub-acromial	3 years	6/ 7/38	3	Imp?

Table I—*Continued*

Case	Name	Sex	Age	Occupation	Diagnosis	Part Affected	Duration	Treatment Began	No.	Result
154	E.G.	F	50	Housewife	Bursitis	Subacromial	2 weeks	5/27/38	6	Imp
155	M.W.	M	55	Physician	Spondylitis Arthritis	Lumbar Sacroiliac	4 weeks	5/28/38	5	Imp
156	P.K.	M	60	Physician	Spondylitis Bursitis	Cervical Calcified subdeltoid	5 months	7/21/38	8	C
157	B.F.	F	55	Housewife	Bursitis	Subacromial	3 months	7/25/38	12	Imp
158	M.M.	M	45	Business	Bursitis	Subdeltoid	2 weeks	8/ 2/38	2	C
159	B.B.	M	54	Physician	Spondylitis Bursitis	Cervical Subdeltoid	8 months	8/ 8/38	5	Imp
160	C.A.	M	43	Antiques	Spondylitis	Cervical	1 month	8/ 8/38	5	Imp
161	S.P.	F	34	Housewife	Spondylitis Bursitis	Cervical Calcified subdeltoid	1 week	8/11/38	2	Imp?

sometimes a slight rise in temperature. These reactions may follow a few hours or a few days after the treatment is started. Sometimes no reaction takes place until the second or third treatment. Some patients experience no ill effects, whereas in others the reactions may be quite severe and may last from one to three days. I have found that the chronic cases are more apt to be followed by reactions than the acute.

With experience it is possible to predict whether a case will respond favorably to the treatment, and one may venture an opinion as to the number of treatments necessary to alleviate the condition. For instance, if a definite subdeltoid bursitis with associated excruciating pain and tenderness is referred for treatment and I find roentgenological evidence of calcification in the region of the bursa, I can say with reasonable certainty that the patient will respond. Again, a case of spondylitis in which there is present lipping of the bodies of the vertebrae with pain and tenderness in this region always yields to high voltage roentgen therapy.

RESULTS

As indicated in the table, the large majority of the patients were completely relieved of pain and remained free of symptoms. Several patients had one or more recurrences and were relieved by a repeated course of treatments. A few did not respond to this form of therapy. A number of cases who were roentgenographed again showed complete absorption of the calcified foci with disappearance of symptoms. For various reasons many of the cases could not be followed up roentgenographically.

REPORT OF CASES

The following are illustrative cases:

Case 2. J. W., male, aged forty-two, was referred by his physician on April 26, 1932. The patient had pain in his right shoulder for several months. For the past few days the pain had become excruciating and radiated down the right upper extremity to the fingers. He was unable to move his arm and could not sleep on account of the pain, which was not affected by morphine. Roentgenographic examination of the right shoulder showed a large calcified subdeltoid bursitis. After one treatment the pain subsided, the patient began to move his arm and slept through the night. A second treatment was given two days later and was followed by complete recovery.

Case 47. J. N., female, aged sixty-five, mother of a physician, is another similar example of calcified subdeltoid bursitis. The patient had pain in the left shoulder for three weeks, which became so agonizing that it could not be controlled by hypnotics. The first treatment relieved the patient and the pain disappeared entirely after three treatments.

Case 129. M. B., male physician, aged fifty, had a calcified subdeltoid bursitis (Fig. 1) which was associated with severe pain. Following treatment the pain disappeared and nine weeks later roentgenographic examination

Fig. 1. Case 129. July 7, 1937. Calcified subdeltoid bursitis.

showed complete absorption of the calcified focus (Fig. 2).

Case 64. L. Z., male, aged forty-five, was referred on April 22, 1935, with a history of pain in the neck, radiating to the left shoulder and down the left arm. This condition had existed for twenty-six days. Diathermy and various other forms of therapy did not relieve the pain. Roentgen examination revealed the presence of a marked spondylitis involving the third, fourth, fifth and sixth cervical vertebrae. After five treatments the patient was free of symptoms.

Case 63. A. W., female, aged fifty. On April 18, 1935, she had been complaining of pain in the anterior subcostal region for more than four years. During this entire period she was never free from pain, was incapacitated and was unable to pursue her occupation. She tried various forms of therapy; spinal fusion was suggested and considered. Roentgen examination revealed the presence of a dorsal spondylitis (hypertrophic osteo-arthritis) with associated lipping of the bodies of the thoracic vertebrae. It was necessary to give eleven treatments before obtaining satisfactory results.

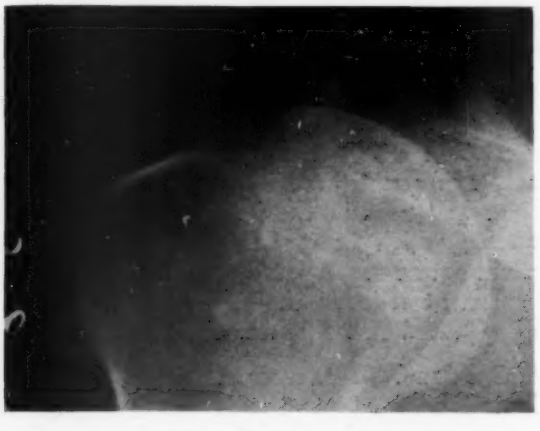

Fig. 2. Case 129. September 8, 1937. After treatment.

Fig. 3. Case 152. April 16, 1938. Calcified subdeltoid bursitis.

Case 141. J. W., male, aged forty-one. The present condition started eight years ago when the patient attempted to lift a rowboat; he experienced a pain in the lumbar region, which persisted. During the past two months the pain became more severe and passed down his right thigh interfering with walking. Roentgen examination showed the presence of a lumbar spondylitis and bilateral sacroiliac arthritis. The patient was given nine treatments and was completely relieved. He had a recurrence

six months later and received seven more treatments with successful results.

Case 21. J. C., female, aged forty, the wife of a physician, had an extraction of an upper molar followed three days later by excruciating neuralgic pain on the same side of the face. The dentist called it a "dry socket" and was unable to control the pain with hypnotics. She received one treatment and was completely relieved. Two days later, following a four hour session in a beauty parlor, the pain recurred.

Fig. 4. Case 152. April 22, 1938. During treatment.

Another treatment was given, followed by complete recovery.

Case 140. B. G., female, aged thirty-two, wife of a physician, was roentgenographed for pain in the left great toe of one year's duration. Only a congenital bifid sesamoid on the plantar surface of the great toe was found. No history of trauma existed. She had great difficulty in walking. During her suffering she had tried various orthopedic shoes and appliances and obtained no relief. Her condition was diagnosed metatarsalgia. Three treatments caused complete and permanent disappearance of pain.

Fig. 5. Case 152. May 4, 1938. During treatment.

She now walks and dances without any difficulty.

Fig. 6. Case 152. May 28, 1938. At end of treatment.

CONCLUSIONS

The cases from which I draw my conclusions were referred by practicing physicians who, as a rule, exhausted all other orthodox and standard forms of treatment before trying roentgen therapy. I had the opportunity to follow up the majority of these patients through the courtesy of their family physicians, some for a period of five or six years. The results obtained certainly justify continuation of this form of therapy in para-arthritis associated with pain. The fact that a scientific basis of roentgen therapy in these conditions is not definitely established is no reason for discarding this form of treatment.

Some of the explanations given for the effect of the treatment are: (1) roentgen rays have an analgesic effect; (2) roentgen rays have a direct effect on lymphocytes and leukocytes and reduce general inflammation and pressure on the nerve sheaths; (3) roentgen rays produce a better blood supply in a diseased area through its effect on the vegetative nervous system with a consequent better tissue metabolism.

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ROENTGEN THERAPY OF BILATERAL PAROTID FISTULA*

By ROBERT J. REEVES, M.D.
DURHAM, NORTH CAROLINA

UNILATERAL salivary fistulae are not uncommon, but bilateral fistulae as a surgical complication are considered quite rare.

Barbera[1] made a critical study of the several operations for salivary fistula and concluded that the suppression of salivary secretions was best produced by high voltage roentgen irradiation, directly on the parotid gland. His technique was as follows: focal distance, 30 cm.; hardness of rays, 180 kv., and filtration of rays through 0.5 mm. copper and 3 mm. of aluminum. The total dosage was 600 roentgens, measured on the skin, given every other day as follows: 300 r followed by 150 r each for the second and third treatment. He found the salivary secretion controlled from the first roentgen treatment. After the third and last treatment, it was definitely suppressed. The fistula was found to heal in two or three days after completion of the treatment. He reported one case in which satisfactory results were obtained by irradiation. Our experience is also limited to one case. Our case did not respond as well as that of Barbera in that it required a larger number of treatments. This may be due to the fact that we were using a shorter wave length but chose to do so because the patient was very fair and the latent skin changes were to be avoided.

CASE REPORT

White female, aged thirteen, came to the hospital July 7, 1936, complaining of bilateral fistulae of eight years' duration.

Past History. Contracted measles at the age of three. The tonsils and adenoids were removed at three and one-half years. On February 26, 1930, at the age of six, she contracted a cold with cough, which lingered in spite of medical attention. A tuberculin test was negative. Six weeks later, the cold was better. The adenoids were again removed. Both ears became abscessed and were opened. The temperature at this time became spiking in character and at times reached 106° F. Blood cultures were positive for hemolytic streptococcus. Roentgenograms of both mastoids showed suspicious evidence of disease and on April 20, the patient was admitted to the Baltimore Ear, Nose, and Throat Hospital, and a bilateral mastoidectomy was performed. The temperature remained spiking in character. The left ankle became swollen and was opened and drained. Repeated blood transfusions were given. The heart at this time showed evidence of endocarditis. The fifth week after mastoidectomy, the left hip was opened and drained. Transfusions were given at intervals of four weeks. Blood cultures during this time became negative. The joints showed less inflammation and cleared up rapidly. The mastoid incisions continued to drain, and in August the patient was discharged from the hospital. At this time, saliva was found coming through the bilateral fistulae. There was a steady flow when eating and a small amount between meals. This drainage has persisted for seven years.

Physical examination at the time of admission was entirely negative except for a small parotid fistula 2 cm. below the middle ear on either side. Marked salivation was noted after chewing gum.

Lipiodol injection disclosed sinus tracts extending into the inferior ducts of both parotid glands.

Roentgen therapy was begun, using 200 kv., Thoraeus filter, and 300 r, measured in air, at two week intervals, to alternate sides. A 3 cm. sq. field was used, shielding the major portion of the parotid gland. A total of 1,800 r was given to each side. Examination on January 9, 1937, showed both sinus tracts closed and no further discharge has been noted.

[1] Barbera, G. Roentgen treatment of salivary fistula. *Policlinico* (sez. prat.), 1936, *43*, 1773–1776.

* From the Roentgen-Ray Department of Duke University Hospital and Medical School.

PERIPHERAL NERVE DESTRUCTION

AN UNUSUAL SEQUEL OF RADIUM THERAPY

By IRVING N. HOLTZMAN, M.D.
Associate Visiting Dermatologist, Brooklyn Cancer Institute

and

WILLIAM E. HOWES, M.D.
Attending Radiation Therapist, Brooklyn Cancer Institute
BROOKLYN, NEW YORK

THE radioresistance of nerve tissue, particularly the fibrils of peripheral nerves, is well known. A review of the literature fails to reveal any report of nerve destruction as a result of irradiation without massive tissue necrosis. In our experience in the treatment of epithelioma of the skin at the Brooklyn Cancer Institute with roentgen rays or radium, we have never encountered a similar instance of nerve destruction following therapy.

CASE REPORT

M. C., aged fifty-eight, married, was admitted on April 27, 1936, with a growth on the left side of the forehead of three months' duration, associated with slight pain and occasional bleeding. She also gave a history of having had a "sore" on her face twenty-three years ago which, she said, was treated by roentgen ray.

Examination on admission revealed an indurated mass 3 cm. in diameter on the left temple with the central portion ulcerated. The left cheek was the site of a pale scarred area,

FIG. 1. Appearance of lesion before therapy.
FIG. 2. Appearance of lesion at present writing.
FIG. 3. Frontal view showing paralysis of left frontalis muscle and edema of periorbital tissue.

apparently healed remnant of the old lesion.

Roentgen examination of the skull on May 19, 1936, showed no bone involvement. Wassermann reaction was negative. Biopsy was reported as basal cell epithelioma.

On May 18, 1936, four platinum needles with filtration of 0.5 mm. were inserted for forty-eight hours with a total dose of 1,500 mc-hr.

On March 1, 1937, lesion was reported as clinically healed.

On May 18, 1937, over the area of the treated lesion, a small subcutaneous nodule 1½ inches lateral to the external canthus was felt. Biopsy of this lesion was reported as basal cell epithelioma, and treatment was again given, on June 3, 1937, with four platinum needles for dosage of 800 mc-hr.

On July 15, 1937, lesion was again described as well healed.

On October 28, 1937, again an acorn-sized hard mass was palpable at the posterosuperior angle of the scar. Roentgenogram of the skull showed no bone involvement.

On November 9, 1937, again four needles were inserted for forty-eight hours with total dosage of 800 mc-hr.

On November 27, patient appeared with the usual marked irradiation reaction over the treated area and also complained of severe pain over left temple and supra-orbital region. The nodule described was no longer palpable. She presented at this time a rather marked edema of the left supra-orbital region, upper and lower left eyelids as well as a paralysis of the left frontalis muscle fibers as evidenced by inability to raise the left eyebrow. There was no disturbance of sensation. This paralysis and edema have persisted up to the present, without any change.

The paralysis came on during the course of the irradiation reaction. The latent period of one week between the application —removal of needles and the onset of paralysis—would rule out any mechanical injury to the nerve by the radium needles. Any puncture would have been followed immediately by paralysis. The fact that pain preceded the onset of the paralysis would indicate an inflammatory change in or around the nerve which, with the appearance of palsy, had apparently progressed to complete interruption. The destruction of the nerve by neoplastic invasion could be ruled out by the disappearance of the nodule treated concomitantly with the onset of paralysis. The edema of the periorbital tissues is attributable to lymphatic obliteration by the irradiation effect. There was no external ulceration at the site of needle application, indicating that no unusual amount of radium had been applied.

The presence of considerable scarring at the site of the lesion from previous therapy might indicate a pre-existing fibrotic band constricting the nerve bundle. The addition of new irradiation reaction with its inflammatory edema may have increased the degree of constriction to final obliteration of the nerve by kinking or stretching; a condition similar to the ulnar nerve paralysis seen as a late sequel of fractures of the lateral epicondyle of the humerus. This late complication, that is secondary obliteration of nerves and lymphatics by scar tissue, should be anticipated in applying intensive therapy to areas containing these structures. We do not believe the paralysis was a direct result of irradiation on nerve tissue itself.

THE USE OF THE CATHODE-RAY OSCILLOGRAPH* FOR MEASURING ROENTGEN TUBE VOLTAGE†

By CHARLES WEYL, S. REID WARREN, JR., *and* DALLETT B. O'NEILL
PHILADELPHIA, PENNSYLVANIA

IT HAS long been recognized that the roentgen tube voltage calibration of roentgenographic and roentgen therapeutic apparatus may be obtained most accurately by means of a high resistance voltage divider and an oscillograph. Formerly, the apparatus required has been expensive and its use has necessitated exposing and processing an oscillographic film for each measurement. Recently, commercial development of inexpensive high value resistors and a cathode-ray oscillograph have made the cost of this apparatus comparable to that of a good sphere gap. This apparatus and its use are described below.

THEORETICAL DISCUSSION

In Figure 1 is shown the schematic wiring diagram of the essential components of this system. Two high resistances R_1 in series with two lower resistances R_2 are connected across the roentgen tube. The midpoint of these series resistances is grounded.

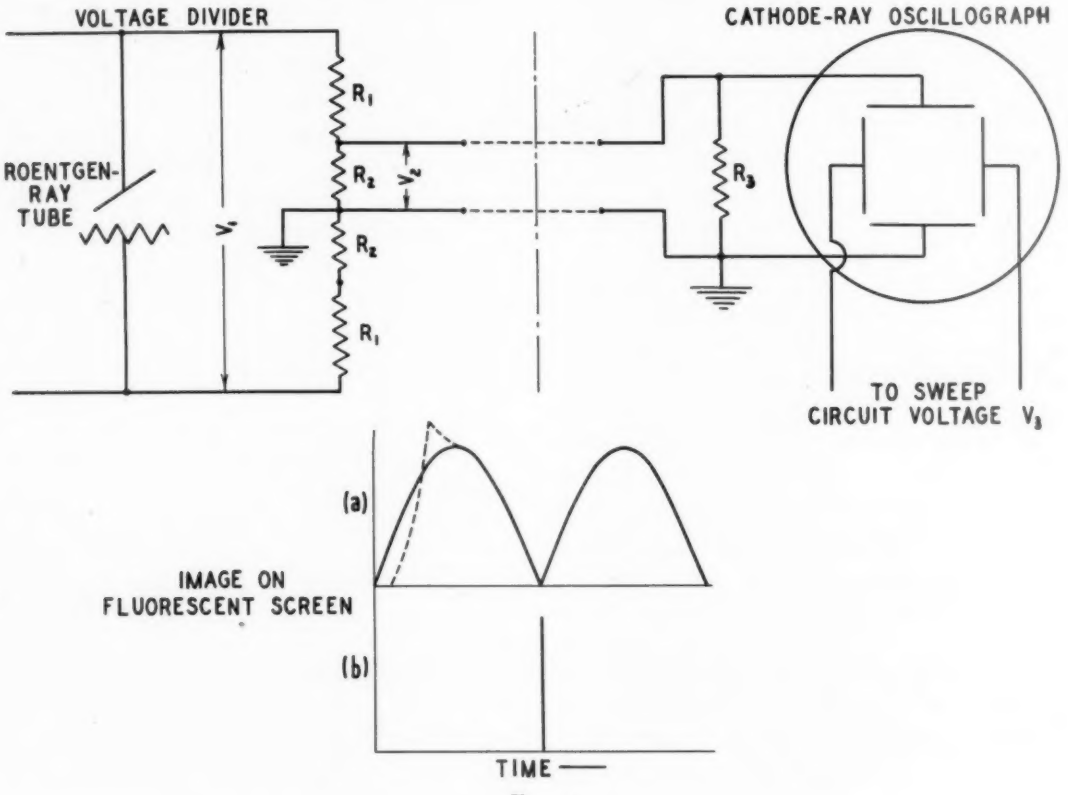

FIG. 1

In this way, a voltage V_2 is obtained across one of the smaller resistances R_2. This voltage V_2 is proportional to the roentgen tube voltage V_1.

The cathode-ray tube shown on the right has a fluorescent screen upon which impinges a beam of high velocity electrons. The image is produced by this beam strik-

* If the instrument is used to observe wave forms visually, it is properly called an *oscilloscope*, but commonly called an *oscillograph*.

† The work described has been conducted at the Moore School X-ray Laboratory of the University of Pennsylvania under grants from L. J. and W. Rosenwald, the University of Pennsylvania, and the National Tuberculosis Association.

ing the screen and the beam is caused to deflect by means of voltages across the two pairs of plates. The voltage V_2 is applied across the vertical deflecting plates, causing the electron beam to move up and down. A separate voltage V_3 is applied to the horizontal deflecting plates. This voltage varies in such a manner that the beam travels across the screen at a uniform velocity from left to right. When it reaches the right hand side it moves back to its starting position instantaneously and repeats its travel. The resistance R_3 shown across the vertical deflecting plates is usually found so connected in commercial cathode-ray oscillographs.

Since the sweep circuit voltage V_3 causes the electron beam to move across the screen uniformly from left to right and the applied voltage V_2 causes it to move vertically at the same time, the resultant image represents the wave form of V_2, as shown by the solid line in Figure 1(*a*). If the period of V_3 is so adjusted that the beam moves across the screen in one cycle of V_2 then the image of each succeeding cycle of V_2 is superposed upon the preceding images. If the oscillograph has been calibrated, the vertical deflection at any instant is a measure of the magnitude of V_2 at that instant. It is then possible to calculate the magnitude of V_1 from the measured value of V_2.

HIGH RESISTANCE VOLTAGE DIVIDER

The high resistance voltage divider used for these measurements must satisfy the following requirements:

(1) It shall impose no appreciable load on the high tension generator.

(2) It shall have low inductance and capacitance so that the wave form will not be distorted. This is particularly important for the observation of transient phenomena.

(3) The values of the resistors must remain constant over long periods of time under the conditions of varying temperature and humidity encountered in roentgen-ray laboratories.

(4) It shall be easily connected to the high tension conductors.

(5) It shall be portable.

Two types of high value resistors have been employed which satisfy these requirements.

The first type of voltage divider was designed for use with roentgenographic apparatus having roentgen tube voltages up to 100 kv. (peak). This voltage divider is shown in Figure 2 (upper). The bank of resistors comprises 100 International Resistance Company Type BT-$\frac{1}{2}$* one megohm units for R_1 and two IRC BT-$\frac{1}{2}$ 100,000 ohm units for R_2. These resistors are mounted as follows: There are six bakelite tubes each 23 inches long and 7/16 inch outside diameter. The inner bore of the tube is sufficiently large to take the BT-$\frac{1}{2}$ units and leave an annular space between the outside of the unit and the inside wall of the order of 1/32 of an inch. Each resistor consists of a piece of glass tube about 0.04 inch in diameter with two No. 20 tinned copper leads attached to the resistance coating of the glass tube; this assembly is then moulded into a cylindrical piece of bakelite in such a manner that the leads extend axially from each end of the cylinder. The one megohm units may be ordered with a tolerance of plus or minus 10 per cent. This reduces the cost of the resistors considerably. When the units are received each unit is measured on a Wheatstone bridge and the 100 units divided into two groups such that the resistance of the first group is equal to 1 per cent or less of the resistance of the second group. One group of resistors is then divided into three parts in the following manner:

(1) Seventeen resistors with a total resistance of approximately 17 megohms;

(2) Seventeen resistors with a total resistance of approximately 17 megohms;

* The apparatus described here and elsewhere in the paper is that which has been used by the authors. It is probable that similar apparatus of other manufacturers can be adapted to perform the same functions.

(3) Sixteen resistors with a total resistance of approximately 16 megohms and one 100,000 ohm unit.

Each of the first two groups of 17 resistors is then arranged in a straight line, the pigtails cut so that their total length is about $\frac{1}{2}$ inch and the whole series soldered together with due care that the total length of the 17 units soldered together does not exceed the length of one of the bakelite tubes. This series of resistors is then slipped into one of the bakelite tubes and a brass piece is fastened to each end of the tube. The lead to each end of the series is soldered to one of these brass fittings. The third tube for this part of the resistance bank is like the other two except that a small hole is made in the tube about $1\frac{1}{2}$ inches from one end in order to bring out the lead from that end of the 100,000 ohm resistor which is connected to one of the 1 megohm resistors. Finally, four pieces of $\frac{3}{4}$ inch square linen base yellow bakelite are assembled to form a square frame, the inside dimensions of which permit the mounting of the six tubes with approximately 4 inches between each two tubes. The frame and the resistors are held together by means of wing nuts. Strips of sheet brass are used to connect the six sections in series. These strips are placed along the bottom surface of the framework to connect sections 1 and 2, sections 3 and 4, and sections 5 and 6. They are placed along the top surface of the framework to connect sections 2 and 3, and sections 4 and 5.

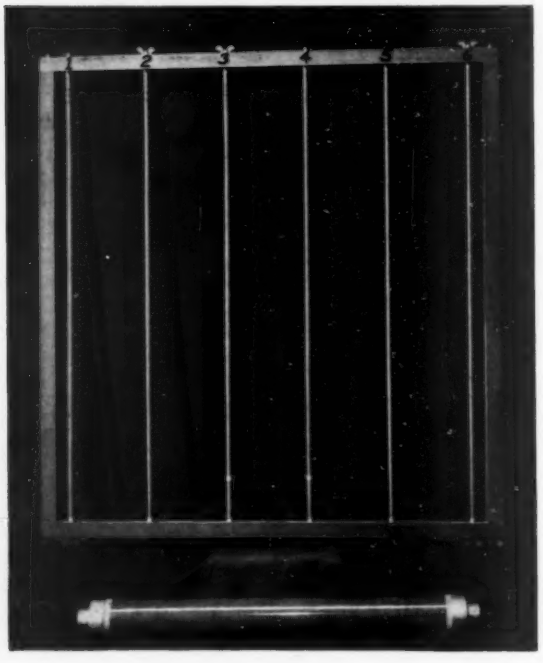

FIG. 2

This voltage divider has been in use for a period of approximately three years. It has been used on short exposures only to prevent overheating of the resistance units. Under these conditions the total resistance of this 100 megohm unit has changed about 2 per cent.

The second type of voltage divider can be used for both roentgenographic and roentgen therapeutic appartaus. It has been used up to voltages of 200 kv. (peak). The lower value resistors R_2 are of the BT-$\frac{1}{2}$ type with resistance of 400,000 ohms each. The two high resistance units are International Resistance Company type MRV and each has a value of 200 megohms. One of these is shown in Figure 2 (lower).

CATHODE-RAY OSCILLOGRAPH

The cathode-ray oscillograph to be used for these measurements must satisfy the following requirements:

(1) The vertical deflection must be proportional to applied voltage.

(2) The fluorescent screen must be of the long persistence type to provide adequate time for observation on short exposures.

(3) The calibration of the vertical deflection must be constant under conditions of line voltage variations of ± 10 per cent.

(4) It must be possible to control the intensity and focusing of the electron beam and the magnitude and frequency of the sweep circuit voltage.

While it is possible to build an oscillograph satisfying these requirements it has been

found advantageous to use one of the commercial models now available. These commercial oscillographs are inexpensive and can be used for other purposes.

The cathode-ray oscillograph which has been used is the RCA type TMV122C which operates on 25 or 60 cycles. If operation on 60 cycles only is necessary type TMV122B may be used. The cathode-ray tube is the RCA type 910 which has a long persistence fluorescent screen. In some oscillographs the leads from the vertical deflecting plates are brought out to pin jacks on the panel. In the particular model used it was necessary to bring these leads out separately. This is easily accomplished by removing the case of the oscillograph and soldering the leads directly to the cathode-ray tube socket terminals. Because of the high voltage of the power supply unit in the oscillograph, it must not be operated until the case has been replaced.

In order to assure constancy of calibration on various supply lines a 60 watt Raytheon type VR2 voltage regulator was used. This voltage regulator operates on a constant frequency (60 cycles/sec.) input voltage from 95 to 130 volts. The oscillograph is operated from the output voltage of the regulator, the rms value of which is 115 volts ±1 per cent. The cathode-ray oscillograph and stabilizer are shown in Figure 3.

METHOD OF USE

The high resistance voltage divider and and cathode-ray oscillograph are connected as shown in Figure 1. A 45 volt battery and a voltmeter are connected across the input to the vertical deflecting plates. The magnitude of the deflection on the screen is measured with a pair of calipers. This permits the calculation of the calibration constant K since $V_2 = Kd$ where d is the deflection of this spot on the fluorescent screen. This relationship should be checked at several values of V_2 to make certain that the deflection is proportional to applied voltage.

The current through R_1 is

$$I = \frac{V_1}{2R_1 + R_2 + \dfrac{R_2R_3}{R_2+R_3}}$$

The same current flows through R_2 and R_3 in parallel and is

$$I = \frac{V_2}{\dfrac{R_2R_3}{R_2+R_3}}$$

Fig. 3

Equating the two values of I there is obtained

$$V_1 = \left(\frac{(2R_1+R_2)(R_2+R_3)}{R_2R_3} + 1 \right) V_2$$

From the calibration of the oscillograph

$$V_1 = \left(\frac{(2R_1+R_2)(R_2+R_3)}{R_2R_3} + 1 \right) Kd$$

To measure the roentgen tube peak voltage the sweep circuit voltage V_3 is reduced to zero. The resulting image during an exposure is a vertical line, as shown in Figure 1(*b*), the height of which may be measured with the calipers, giving a value for d. The exposure time for this measurement should be 1/20 second or more to provide sufficient brightness of the image. If the measurement is made in a room having low illumination or with a hood over the front of the cathode-ray oscillograph the image will persist for thirty to sixty seconds.

In order to observe the wave form of the roentgen tube voltage the magnitude of V_3 is adjusted to provide a horizontal deflection just slightly less than the width of the cathode-ray tube. The period of V_3 is adjusted to move the spot across the screen once every cycle of V_2. In this way, each cycle of V_2 is superposed and a bright image of the wave form will result. If the timer on short exposures does not start or end on zero there will be noted on the screen images which do not coincide with the normal wave form as shown by the dotted line in Figure 1(*a*).

In order to use the cathode-ray oscillograph for checking the exposure time the period of V_3 is increased so that the spot moves across the screen in a longer time than is produced by the timer. Each half-cycle of roentgen tube voltage may then be seen separately and counted. It is necessary that the sweep period be greater than the exposure time to avoid any superposition of half-cycle images.

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Twenty-fifth Annual Meeting: New York City, June 10–11, 1940.

EDITORIALS

AIR INJECTION IN THE ROENTGEN DIAGNOSIS OF CORTICAL AND MEDULLARY TUMORS

With the widespread use of the Potter-Bucky diaphragm as an aid in roentgen diagnostic procedures, the employment of gases as contrast media in the peritoneal cavity has fallen somewhat into disuse though in selected cases they still have a very distinct and definite place. Shortly after the announcement of the use of gases in the peritoneal cavity as contrast media, a number of observers independently called attention to the improved visualization of the kidney by the retroperitoneal injection of oxygen or carbon dioxide and this method of examination of the perirenal space assumed very shortly considerable diagnostic importance. While most observers called attention to the ease with which the adrenal glands might be visualized by this procedure, it was not until Mosenthal and Löser and Israël emphasized the importance of perirenal retroperitoneal insufflation of air as an aid in the diangosis of enlargement of the adrenal glands that there was a revival of interest in this procedure. This revival of interest came at the time when the adrenal glands were being investigated as an etiological factor in some of the obscure clinical syndromes.

Quite recently Cope and Schatzki[1] have called attention to the importance of perirenal retroperitoneal insufflation of air as a diagnostic aid in tumors of the adrenal glands. Their study is based on 163 separate injections of air in 78 patients. In all of their cases studied there was some clinical reason to suspect disease of the adrenal glands. The size of the shadows outlined by air visualized on the roentgen film was confirmed by operative exposure through a retroperitoneal approach in 15 patients and by postmortem examination in two patients.

A great deal of the success of the injection of air into the perirenal space is due to the technique of the procedure, and Cope and Schatzki have given in detail the technique which they have followed in all of the cases studied. The site of choice for injection of air into the perirenal space is the triangular fat pad below the kidney. The lower pole of the kidney rests in a mass of fat which lies between the anterior and posterior folds of renal fascia above the pelvic brim. The presence of a ptotic kidney should first be excluded by palpation or by roentgen examination. They call attention to the meticulousness with which the technique should be followed in order that the best results may be achieved and also to obviate the occurrence of any untoward symptoms.

Not only the site of the injection but the depth to which the needle passes and the volume of the injection together with the rate of injection and the pressure used are of importance in the success of the procedure.

Cope and Schatzki call attention to the technique which they used in avoiding certain possible complications, namely, air embolism, and the possibility of the insertion of the needle into the kidney substance producing a hematoma of the renal capsule and also the avoidance of passing the needle into the adrenal gland itself thereby producing a hematoma of this gland. The danger of these complications is considerably lessened by introducing the needle at the site as selected by Cope and Schatzki,

[1] Cope, Oliver, and Schatzki, Richard. Tumors of the adrenal glands. I. A modified air injection roentgen technic for demonstrating cortical and medullary tumors. *Arch. Int. Med.*, 1939, *64*, 1222–1238.

namely the lower rather than the upper portion of the renal space. With the low pressure and the slow rate of injection of the air pain is minimized. Following the injection of the air the patient is asked to walk around for at least half an hour. They have found it unnecessary in their technique to massage the loin. In the majority of cases a complete diffusion of the air and better visualization of the organs were obtained after twenty-four hours. It is their practice to take one set of films within an hour after the injection and a second set twenty-four hours later; anteroposterior and lateral views are taken at each time. It is unnecessary to have any special preparation of the gastrointestinal tract except that the colon should be clean. They have found it advantageous in a study of the lateral films to inject the air on one side at a time and they have allowed a week to elapse between injections. In this length of time practically all of the air previously injected has been absorbed. In their study they found that the normal adrenal gland is not always visualized on the roentgenograms after the injection of air; however, when outlined in its variety of shapes it conforms closely to the gross anatomy of the adrenal gland as seen at operation or autopsy. The left gland is generally elongated; the right, pyramidal.

One difficulty of interpretation is caused by superimposed irregular shadows cast by the fat and connective tissue surrounding the gland. The density of the shadow cast by the normal adrenal gland is ordinarily greater than that cast by surrounding tissues but is of less density than the upper pole of the kidney.

In their experience the anteroposterior films give the most satisfactory results and they have in some of their studies used intravenous pyelographic media simultaneously with the air injection.

This method of examination is of considerable importance in the visualization of small tumors not shown by other means and it is is also of value in the exclusion of the adrenal glands as the primary source of disease by demonstrating normal gland shadows. It is an aid in demonstrating bilateral hyperplasia of the adrenal glands and in the differential diagnosis of shadows in the perirenal area which have been mistaken for tumors of the adrenal glands.

In commenting upon the usefulness of such a procedure, Cope and Schatzki call attention to the dangers which may follow the injection of air into the perirenal spaces or into the body cavities, but with the safeguards which they have outlined they believe it is a method which can be used on an ambulatory patient. In the 163 separate injections there have been no fatalities and no signs of air embolism. The only complications in their series occurred in the first twenty cases in which the site of injection recommended in the literature was used. They emphasize an important point in their technique, namely, that there is such a variation in the anatomy of the individual patients that no actual measurements so far as depth to insert the needle is concerned can be followed. One must perforce have surgical judgment in utilizing such a procedure together with a complete knowledge of the anatomy and above all the position of the needle itself must be carefully checked before air is injected.

In spite of safeguards, perirenal insufflation is not recommended without reservation and it should be used only in those cases in which there is a definite suspicion of disease of the adrenal glands or tumors in that area. The danger of the method is also noted in a recent report by Weyrauch[2] of a case in which death occurred from air embolism following perirenal insufflation.

The modified technique of Cope and Schatzki tends to lessen the dangers in the employment of this method for the diagnosis of perirenal tumors and at the same time adds tremendously to our diagnostic armamentarium by enabling us to recognize these tumors earlier in the course of the disease and in certain cases to obviate the necessity of surgical exploration.

[2] Weyrauch, Henry M., Jr. Death from air embolism following perirenal insufflation. *J. Am. M. Ass.*, Feb. 24, 1940, *114*, 652–653.

HOWARD EDWIN RUGGLES
1886-1939

AT THE height of his meteoric career on December 28, 1939, in his country home near San Francisco, there quietly passed to his rest the "Dean" of Roentgenology of the Pacific Coast, Howard Edwin Ruggles.

He was born in San Francisco and received his Bachelor Degree from Stanford University in 1907 and his Medical Degree from Harvard Medical School. He returned to San Francisco in 1913 and spent the rest of his life in that city except for the time he so ably served his country as Captain attached to Base Hospital No. 30 during the World War. His first appointment of roentgenologist to St. Luke's Hospital was

quickly followed by an appointment to the Staff of the University of California Medical School where he rose to the rank of Clinical Professor and head of the Department of Roentgenology. At the time of his death, besides the above mentioned appointments he was Chief Consultant to the Southern Pacific Hospital, Shriners Hospital, Children's Hospital, and the University of California Service at the San Francisco Hospital.

In 1918 he collaborated with his beloved friend and teacher, George Holmes, of Harvard in the publication of a text in roentgenology which has had many editions and is now a standard text for most colleges in America as well as abroad.

In 1936 he collaborated with Miley Wesson in a book on urological diagnosis.

He was a member of the San Francisco County Medical Society, California State Medical Society, the American Medical Association, and of the American Roentgen Ray Society which he loyally served as Vice President, member of the Executive Council, and at the time of his death was Chairman of the Publication Committee.

With a rapier-like mind, skillful hands, and a dynamic personality, no man has had a greater influence on the advancement of roentgenology than Howard Ruggles.

He was a master technician and was never content with anything but the best in the diagnostic value of his roentgenograms. His diagnostic ability was uncanny, swift, and sure, and as a teacher he was unexcelled. The first practical tilting fluoroscope and the Ruggles Sinus Device were children of his brain and he developed one of the earliest methods of successful moving roentgenograms of the heart and that in the days of inefficient tubes, slow screens, and films.

He cared little for competitive games and his only interest in sports was in backing the football team of his alma mater, Stanford, and this he did most strenuously.

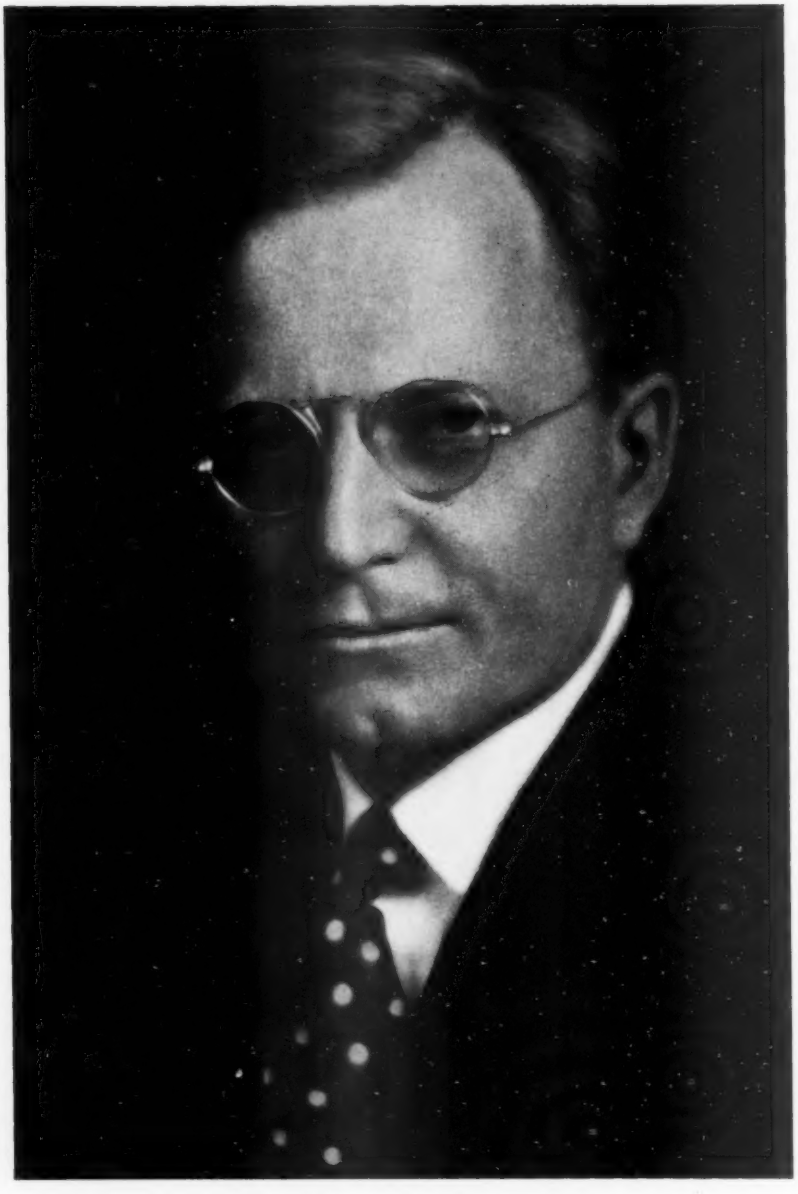
His principal hobby was photography and his collection of moving pictures in color of the South Seas excel the professional travelogues and were a source of delight to his many friends.

He was always most generous in his aid to the younger medical men and gave many a young roentgenologist his first step up the ladder of success and throughout our land are many prominent roentgenologists who are indebted to Howard Ruggles and to them he is still the "Chief."

The high plane that roentgenology holds in the medical world, particularly on the Pacific Coast, is due in great measure to the inspiration, patience, and untiring energy of Howard.

He lived not in vain. Because of him our profession and our lives are so much richer and fuller that we can have faith that another such as he may come.

Lloyd Bryan



LOUIS S. WEAVER
1877-1940

IN A world where men have done much and have much yet to do, we pause at times to render praise to those who have led in these accomplishments. Today we pause in memory of Dr. Louis S. Weaver, truly a man who accomplished much of work and play and service during his short span of life. The many types of his services are revealed by his activities. For many years he served on the York Hospital Staff, being Chief Surgeon for the past eight years. He was a charter member of the American Roentgen Ray Society; a member of the American College of Surgeons; past president of the York County Medical Society; past president of the York Medical

Club; past president of the York Lions Club. He was a member of the York Recreation Board; of the York Welfare Board; the York Chamber of Commerce and of the Boy Scout Council. He served six years as a City School Director. A veteran of the Spanish American War and of the World War, he served as Commander of the York Post No. 127, American Legion. He was a regular attendant at Church. He was a trustee of Gettysburg College; and a charter member of his college fraternity, Phi Kappa Psi, in which he was intensely interested. An athlete, he was once City tennis champion.

He worked hard, he played hard. His honesty and his steadfast support of his friends characterized this man. With him, a proposition was either right or it was wrong; one always knew where he stood on all matters requiring a decision. Such an attitude required courage, but brought inward peace. He had a zest for work but did not make gain his god; he remembered the contributions of the past in science and in progress, but he was not enslaved by tradition. He had religious interests, without becoming too far removed from the practical world. His surgical skill, technique, and endurance were recognized by all who knew him.

He leaves three children, Kathryn, Frank and Louis. His wife, Romaine, preceded him in death several years ago.

We shall miss his cheerful manner, his judgment, and his surgical skill. His place will be hard to fill. Our profession truly has lost one of its most distinguished men.

L. S. Landes

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. C. B. Peirce, Royal Victoria Hospital, Montreal, Canada. Annual Meeting: Hotel Statler, Boston, Mass., Oct. 1-4, 1940.

American College of Radiology
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill. Next Annual Meeting: Commodore Hotel, New York City, June 12, 1940.

Section on Radiology, American Medical Association
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: New York City, June 10–14, 1940.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting, 1940: To be announced.

Radiological Section, Baltimore City Medical Society
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets twice annually in May and September.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill. Next meeting Peoria, Ill., May 21–23, 1940.

Radiological Section, Los Angeles Co. Med. Soc.
Secretary, Dr. Wilbur Bailey, 2007 Wilshire Blvd., Los Angeles, Calif. Meets on second Wednesday of each month at County Society Building.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Brooklyn Roentgen Ray Society
Secretary, Dr. L. J. Taormina, 1093 Gates Ave., Brooklyn, N. Y. Meets monthly on first Tuesday, October to April.

Buffalo Radiological Society
Secretary-Treasurer, Dr. Joseph S. Gian-Franceschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months, place of meeting selected by the host.

Chicago Roentgen Society
Secretary, Dr. C. J. Challenger, 3117 Logan Blvd. Meets second Thursday of each month October to May inclusive at the Hotel Sherman.

Cincinnati Radiological Society
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. H. A. Mahrer, 10515 Carnegie Ave. Meets at 6:30 P.M. at Mid-Day Club rooms on fourth Monday each month, October to April, inclusive.

Denver Radiological Club
Secretary, Dr. P. R. Weeks, 520 Republic Bldg., Denver, Colo. Meets third Friday of each month.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

Florida State Radiological Society
Secretary, Dr. J. N. Moore, 210 Professional Bldg., Ocala, Florida. Meetings in May and November.

Georgia Radiological Society
Secretary, Dr. R. C. Pendergrass, Prather Clinic Bldg., Americus, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meetings held quarterly, on the fourth Sunday of the month.

Indiana Roentgen Society
Secretary, Dr. C. C. Taylor, 23 E. Ohio St., Indianapolis, Ind. Meeting held the second Sunday in May annually.

Kentucky Radiological Society
Secretary, Dr. J. C. Bell, 402 Heyburn Bldg., Louisville. Meets annually in Louisville on third Sunday afternoon in April.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

Michigan Association of Roentgenologists
Secretary, Dr. C. S. Davenport, St. Lawrence Hospital, Lansing. Three meetings a year, Fall, Winter, Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. I. I. Cowan, Mt. Sinai Hospital, Milwaukee, Wis. Meets monthly on first Friday at University Club.

Minnesota Radiological Society
Secretary, Dr. J. P. Medelman, 572 Lowry Medical Arts Bldg., St. Paul.

Nebraska Radiological Society
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M., at either Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. A. O. Hampton, Massachusetts General Hospital, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

Radiological Society of New Jersey
Secretary, Dr. W. J. Marquis, 198 Clinton Ave, Newark. Meets annually at time and place of State Medical Society. Mid-year meetings at place designated by president.

New York Roentgen Society
Secretary, Dr. R. D. Duckworth, 170 Maple Ave., White Plains, N. Y. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

North Carolina Roentgen Ray Society
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year—January, May, November.

Pacific Roentgen Club
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport, Pa. Next annual meeting, Hershey Hotel, Hershey, Pa., May 17–18, 1940.

Philadelphia Roentgen Ray Society
Secretary, Dr. B. R. Young, Temple University Hospital. Meeting first Thursday of each month from October to May inclusive, at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. H. W. Jacox, 4800 Friendship Ave., Meetings held second Wednesday each month, 4:30 P.M., October to June at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. S. C. Davidson, 277 Alexander St., Meets on second Thursday from October to May, inclusive, 8 P.M., Rochester Academy of Medicine Building.

St. Louis Society of Radiologists
Secretary, Dr. W. K. Mueller, University Club Bldg. Meets fourth Wednesday of October, January, March and May, at a place designated by the president.

San Francisco Radiological Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco. Meets monthly on first Monday at 7:45 P.M., alternately at Toland Hall and Lane Hall.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

South Carolina X-Ray Society
Secretary, Dr. Hillyer Rudisill, Jr., Roper Hospital, Charleston. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. F. B. Bogart, 311 Medical Arts Bldg. Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. L. W. Baird, Scott and White Hospital, Temple, Texas. Next annual meeting, January 18, 1941, Sherman, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. V. W. Archer, University Hospital, University, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. K. J. Holtz, American Bank Bldg., Seattle. Meets fourth Monday of each month at the College Club, Seattle.

Cuba

Sociedad Cubana de Radiologia y Fisioterapia
Secretary, Dr. Francisco Padron, Enrique, Villuendas 64, Havana, Cuba. Meets monthly in Havana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Meets monthly on third Thursday, from November to June inclusive, at 8:15 P.M., 32 Welbeck St., London.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month during the winter at 8:15 P.M. at the Royal Society of Medicine, 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. Barbara M. Key, 32 Welbeck St., London, W.1, England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to Nov. incl. for scientific discussion.

Canadian Association of Radiologists
Secretary, Dr. A. C. Singleton, Medical Arts Bldg., Toronto, 5, Ontario.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext., Halifax, N.S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

Continental Europe

Belgian Society of Roentgenology
Secretary, Dr. J. Boine, Avenue des Ailiés, 134, Louvain (Belgium).
Meets monthly on second Sunday at d'Egmonds Palace, Brussels, except in the summertime.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7, Madrid, Spain. Meets monthly in Madrid.

Société de Radiologie Médicale de France
Meets monthly on second Tuesday, except during months of August and September, 12 Rue de Seine, Paris.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean, La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse, Biel. Meets annually in different cities.

Société Francaise d'Electrothérapie et de Radiologie Médicale
Meets monthly on fourth Tuesday, except during month of August and September, 12 Rue de Seine, Paris.

Association of German Roentgenologists and Radiologists in Czecho-Slovakia
Secretary, Dr. Walter Altschul, German University, Prague, II.52.

Deutsche Röntgen-Gesellschaft (Gesellschaft für Röntgenkunde und Strahlenforschung)
Meets annually in April, alternating one year in Berlin, one year in some other German city. Meets in addition every two years with the Gesellschaft deutscher Naturforscher und Aerzte.
Permanent Secretary, Professor Dr. Haenisch, Klopstockstrasse 10, Hamburg, Germany.

Süd- und Westdeutsche Röntgensellschaft
Meets annually in different cities.

Nord- und Ostdeutsche Röntgengesellschaft
Meets annually in different cities.

Dutch Society of Electrology and Roentgenology
Holds two meetings a year in Amsterdam, one in the Spring, and one in the fall.

Societa Italiana Radiologia Medica
Secretary, M. Ponzio, University of Turin, Prof. Turin

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania.
Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly on first Monday at 8 o'clock.

Polish Society of Radiology
Secretary, Dr. Jan Kochanowski, 45 Gornoslazka St., Warsaw. Meets annually.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. B. Krynski, 11 Zielna St.
Meets once a month except in the summertime.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association. Each of the following societies, with exception of the Denmark Society, meets every second month except in the summertime:

Society of Medical Radiology in Sweden
Meets in Stockholm.

Society of Medical Radiology in Norway
Meets in Oslo.

Society of Medical Radiology in Denmark
Secretary, Dr. G. Biering, Copenhagen.
Meets the second Wednesday of each month from October to July in Copenhagen, at 8 o'clock in the State Institute of Roentgenology.

Society of Medical Radiology in Finland
Meets in Helsingfors.

Vienna Roentgen Society
Meets first Wednesday of each month, at 6:30 P.M. at Zentral-Röntgen Institut des allgemeinen Krankenhauses Alserstrasse 4.

Orient

Japan X-Ray Association
c/o Orthopedic Surgery, Tokyo Imperial University. Meets annually in April.

Kinki Roentgen-Abend Society
Director, Dr. Prof. Taiga Saito, Ogawaoike Tyoto Japan. Meets bi-monthly on third Sunday.

BULLETIN OF THE INTER-SOCIETY COMMITTEE FOR RADIOLOGY

THE MODERN HOSPITAL AND ITS RELATION TO THE PRACTICE OF MEDICINE

During the past twenty or thirty years there have been rapid and extensive changes in the traditional function of the hospital in its relation to the practice of medicine and the delivery of medical care to the American people.

A much larger portion of sick patients are cared for in the private general hospital today than was the case a generation ago. Out of every dollar spent for medical care in this country approximately twenty-four cents goes to pay a hospital bill, either in private or government hospitals.[1] This represents a cost of about 656 million dollars per year, of which 300 million is provided by taxes, 302 million by patients' fees, and the remainder by contributions and endowments.[2] The cost of hospitalization as a result of illness is almost as high as the total cost for private physicians' services during a given year. About 86 per cent of the total families in the United States require the services of a physician in an average year but only 20 per cent of the total have at least one hospital case.[3] Less than 50 per cent of all family illnesses are hospitalized, yet the costs for hospitalization alone in these cases equals the entire expenditure for all illnesses of the entire family in an average year.[4] Half the family bills for illnesses goes for hospital care, but in any one year only one person in fifteen has hospital care.[5]

The cost of medical care in America, therefore, is very largely a cost for hospital care. That this latter cost is entirely justified is a matter open to serious question. A number of factors has contributed to the increased use of hospitalization and the consequent increased cost of sickness. Many qualified students of the question declare that there is too much hospitalization, and that many patients subjected to the expensive facilities of the hospital could well be cared for in their own home. Three factors are responsible for this alleged over-hospitalization. Doctors have referred their patients to the hospital because it was more convenient for the doctor to render his services there, patients have demanded that they be admitted to the hospital when they could just as easily have been treated in their homes, and hospitals have encouraged the use of their facilities with an occasional disregard of the need of the patient for those facilities.

Whether deliberately or not, current tendencies on the part of hospitals find their keynote in the first recommendation made by the Committee on the Costs of Medical Care. In 1929 the Committee recommended that medical care be furnished by groups, organized around a hospital.[6] Thus the hospital becomes the dominant factor in the delivery of medical services. The importance of the traditional family doctor as the prime figure in an adequate health program is obscured in a trend toward institutionalized medicine with the hospital as the central figure.

Steps toward this goal were made when hospitals began the operation of pay clinics and out-patient departments in competition with the private physician's office. Abandoned was the old concept that out-patient services and part-pay clinics should be maintained only in teaching institutions where patients should be used for clinical material. Further steps were taken when the "middle rate" plan was adopted by a number of hospitals, in which patients were admitted on an adjusted fee basis including not only the hospital bill, but the bill for medical care as well. Other hospitals instituted "flat rate" or "all inclusive" plans under which a patient could

[1] Medical Care for the American People, Committee on the Costs of Medical Care, p. 15.
[2] Economic Problems of Medicine, Christie, p. 61.
[3] Economic Aspects of Medical Services, University of Chicago Press, p. 6.
[4] Fundamental Facts on the Costs of Medical Care, Falk, p. 13.
[5] Doctors, Dollars and Disease, Foster, p. 15.
[6] Medical Care for the American People, Committee on the Costs of Medical Care, p. 109.

be admitted to the hospital for diagnostic services and pay a flat fee covering the complete cost for medical services and hospital facilities. Still further and more radical steps were taken when hospitals began the sale of insurance plans which offered as benefits not only the use of the physical facilities of the hospital, but the services of medical specialists as well.

Income from endowments and contributions was sharply curtailed after 1929. The 7,000 hospitals in this country represent a capital investment of more than 3 billion dollars, with about 22 million added each year for expansion[7] and the interest on these investments plus other fixed charges created a difficult problem in finance. Hospitals began to cast about for some new source of revenue to take the place of decreased endowment income.

Group hospitalization insurance presented itself as a happy solution for the problem, so far as hospitals were concerned. Started as an experiment by one hospital in Dallas in 1927, the idea gained little attention until hospitals began to feel the pinch of depression. Then it began a wide and rapid growth.

Less than 65 per cent of the beds in nongovernmental hospitals are normally occupied,[8] yet fixed charges remain about the same regardless of the number of occupants. So even if hospital insurance increased the number of patients, the steady annual income from insurance premiums would provide the new and necessary revenue.

If hospitals had stopped with the establishment of insurance plans for hospitalization there would have been little objection from the medical profession which has guarded so zealously the traditional system of American medicine. But, unfortunately, a number of hospitals went still farther to secure new revenue. Ostensibly operated on a non-profit basis, they began to adopt the rôle of a middle-man in the delivery of certain medical services to realize a profit on the services of physicians. Out-patient clinics for pay patients began to grow. Hospitals began to compete with private physicians' offices. Pathologists and radiologists working in the hospital found themselves competing with themselves in their private offices and with their colleagues. Frequently the department of radiology or pathology was operated in such a manner that a large portion of the fees patients paid for these medical services were diverted into the hospital treasury to meet the interest on building investments and other charges.

Here marks a radical departure in the former position of the private non-profit hospital in the medical picture. Doctors had always regarded the hospital as a specially equipped hotel where patients, who could not be satisfactorily treated in their own home, could be hospitalized. Now the hospital was beginning an invasion of the medical field, and assuming the position of a commercial entrepreneur by interjecting itself between a doctor and his patient, demanding its portion of the fees passing between the patient and his doctor. Many hospitals collected premiums under insurance contracts providing not only hospitalization but the services of physician, radiologists and pathologists as well.

So the lines of difference were clearly drawn. The medical profession has come to the realization that further encroachments on all types of medical practice are almost sure to follow unless hospitals can be prevailed upon to abandon this unfortunate course, return to the business of hospitalization, and leave the practice of medicine to private physicians.

The medical profession has long maintained that the institutionalization of medical practice or the application of the principles of socialization would result in a lowering of the quality of medical care available to the American people. Taking cognizance of the gradual trend toward such a condition through encroachment of hospitals upon the field of private medical

[7] Economic Problems of Medicine, Christie, p. 60.

[8] Medical Care for the American People, Committee on the Costs of Medical Care, p. 26.

practice, the Judicial Council of the American Medical Association at the annual meeting in 1936, issued a strong statement to the effect that:

> . . . It would seem that in this time of extensive changes in hospital economics the point had arrived at which further marriages between hospitals and staff physicians that make the doctor of medicine the servant of the hospital should be stopped and a series of attempts at divorce among marriages that have already taken place should be instituted. Our accepted ethical principles are adequate at the present time and Hospitals would be of invaluable assistance. It is not an impossible task but will need a militant local and national ethical spirit behind it and a frowning on those individuals in the profession who on personal grounds do not object to the gradual subjugation of the medical profession in the growth of hospital domination.[9]

The Code of Ethics of the American Medical Association provides that:

> It is unprofessional for a physician to dispose of his professional attainments or services to any lay body, organization, group of individuals, by whatever name called, or however organized, under terms or conditions which permit a direct profit from the fees, salary or compensation received to accrue to the lay body or individual employing him. Such a procedure is beneath the dignity of professional practice, is unfair competition with the profession at large, is harmful alike to the profession of medicine and the welfare of the people, and is against sound public policy.[10]

In this manner the conflicting viewpoints of the medical profession and the hospital group is defined.

The fears of the medical profession in this regard are fully justified by recent statements appearing in hospital publications. These statements evidence a complete disregard of the desires and recommendations of the medical profession. Repeatedly, statements have appeared that hospital service includes radiological, anesthesiological and laboratory services; that the hospital depends upon the profits from the operation of certain medical departments to maintain the hospital and increase its income; that the services of the clinician, the surgeon, the pathologist and the radiologist, as well as the nurse, are a part of the hospital and are services to be provided by the hospital free from the dictation of medical men.

Practically all States in the Union have enacted statutes prohibiting the practice of medicine by corporations. Similar statutes prevail prohibiting the corporate practice of law, dentistry, and other learned professions. These laws have been passed for the simple reason that the practice of the learned professions by artificial legal entities would not be to the best interest or welfare of the people. Hospital corporations are artificial entities and it is hard to understand why the same reasoning should not be applied in their case. The public would surely suffer as the result of this kind of corporate practice the same as it would in the case of non-hospital corporations. The type of organization is relatively immaterial. In any case the traditional and indispensable personal relationship between a doctor and his patient will be destroyed or injured under such conditions.

It is true that the authority of the common law is not clear upon the question of the corporate practice of medicine by hospitals. Although important decisions have been rendered by the Supreme Court in several states upholding the statute prohibiting corporate practice, there seems to be a universal reluctance to apply such rulings to hospital corporations. The law, however, has traditionally lagged at least a generation behind the sciences, and the question involved here is a scientific one and not a legal one. The important point to consider is whether the science of medicine will be preserved and advanced. The general opinion among medical men is that corporate practice, either by hospitals or any other kind of corporation, will hinder the progress of the science and practice of medicine.

The medical profession has found it nec-

[9] *J. Am. M. Ass.*, 1936, *106*, 1197.

[10] Principles of Medical Ethics, American Medical Association, p. 20.

essary to wage a militant battle against various forces which have during recent years attempted to override the opinion of medical men and bring about revolutionary changes in the form of medical practice in America. In the past the medical profession has looked upon the hospitals as its partner and ally in a relentless fight to preserve the practice of medicine from prejudicial influences.

It will be a great misfortune if the unselfish demands of the medical profession, standing on a firm realization of idealistic principles and scientific truths, be disregarded by a former ally and friend and the hospital shall be found on the side of those dangerous agencies which would substitute their opinions regarding matters of health for the opinions of the doctor.

The threat of hospitals in invading the field of medicine, upsetting the traditional relationships between physician and patient, and supplanting free private enterprise with regimented institutionalization is no less than the threat of other radical forces which the hospitals themselves have denounced.

Mac F. Cahal

AMERICAN BOARD OF RADIOLOGY

The next examination to be conducted by The American Board of Radiology will be in New York City, June 7–9, 1940, but the quota is practically filled for this examination. We would therefore urge those who are anxious to be examined this year to send their applications in immediately so that they may be scheduled for the examination to be conducted in Boston, Massachusetts, September 27–29, 1940.

Address all communications to: Dr. B. R. Kirklin, Secretary, The American Board of Radiology, Mayo Clinic, Rochester, Minnesota.

Following is a list of those who have been certified or granted additional certification during 1939:

1. Abrams, Hyman S., Tuscaloosa, Ala., Radiology
2. Ackermann, Alfred J., Oklahoma City, Okla., Roentgenology.
3. Algin, Sergius, Indiana, Pa., Roentgenology
4. Allen, William E., Jr., St. Louis Mo., Radiology
5. Almy, Max A., Rochester, N. Y., Roentgenology
6. Andrew, F. D., Rochester, N. Y., Radiology
7. Ansprenger, Aloys G., Macomb, Ill., Radiology
8. Athle, Laxman H., Bombay, India, Radiology
9. Bachman, Arnold L., Stamford, Conn., Radiology
11. Barner, John L., Cincinnati, O., Therapeutic Radiology
10. Baltimore, Louis, Los Angeles, Cal., Roentgenology
12. Bayliss, Jacob W., Buffalo, N. Y., Roentgenology
13. Beatty, Samuel R., Denver, Colo., Radiology
14. Bell, Charles E., East St. Louis, Ill., Radiology
15. Bernstein, Alfred J., New York, N. Y., Radiology
16. Bersack, Solomon R., Springfield, Mass., Radiology
17. Bond, Thomas B., Fort Worth, Texas, Roentgenology
18. Bonis, Alexander, New York, N. Y., Radiology
19. Boswell, Frederick, P., Montgomery, Ala., Radiology
20. Bracher, George J., Seattle, Wash., Diagnostic Roentgenology
21. Brindle, Harry R., Asbury Park, N. J., Roentgenology
22. Brown, William L., Sr., Chicago, Ill., Radium Therapy
23. Bryan, William W., Montreal, Canada, Radiology
24. Bugbee, Edwin P., Philadelphia, Pa., Radiology
25. Burvill-Holmes, E., Philadelphia, Pa., Radiology
26. Butler, William J., Providence, R. I., Radiology
27. Carr, Gladys L., Hempstead, N. Y., Roentgenology
28. Carroll, William J., Hot Springs, Ark., Radiology
29. Casper, Stephen L., Philadelphia, Pa., Radiology
30. Caulk, Ralph M., Washington, D. C., Radiology
31. Chang, C. William, New York, N. Y., Radiology
32. Cohen, Paul W., New York, N. Y., Therapeutic Radiology
33. Colton, Benjamin, Flushing, N. Y., Diagnostic Roentgenology
34. Conahan, Thomas J., Hazleton, Pa., Radiology
35. Cooney, James P., Washington, D. C., Radiology
36. Cooper, George, Jr., University, Va., Roentgenology
37. Cooper, Robert W., Shreveport, La., Radiology
38. Curtzwiler, Francis, C., Toledo, O., Radiology
39. Daversa, Joseph J., Brooklyn, N. Y., Roentgenology
40. Dell, J. Maxey, Gainesville, Fla., Roentgenology
41. Doble, Eugene H., Presque Isle, Me., Diagnostic Roentgenology
42. Dowdy, Andrew H., Rochester, N. Y., Radiology
43. Echternacht, Arthur P., Indianapolis, Ind., Radiology
44. Elliott, William J., Worcester, Mass., Radiology
45. Epstein, Bernard S., Brooklyn, N. Y., Radiology
46. Euphrat, Edwin J., Elkins, West Va., Radiology
47. Evans, William A., Jr., Detroit, Mich., Radiology
48. Fierstein, Jacob, New York, N. Y., Roentgenology
49. Flanagan, E. Latane, Richmond, Va., Roentgenology
50. Gartenlaub, Charles, New York, N. Y., Roentgenology
51. Gilbert, Philip D., Camden, N. J., Radiology
52. Gilmore, John H., Chicago, Ill., Radiology
53. Good, C. Allen, Rochester, Minn., Radiology
54. Grady, Mary Alice, San Francisco, Cal., Roentgenology
55. Gray, Charles M., Tampa, Fla., Radiology
56. Grimm, Homer W., Pittsburgh, Pa., Radiology
57. Hall, James B., Johnstown, Pa., Roentgenology
58. Hankins, Walter D., Johnson City, Tenn., Roentgenology
59. Hanley, D. Reed, Streator, Ill., Roentgenology
60. Hartung, Walter, Iowa City, Ia., Radiology
61. Harwell, Wilbur R., Shreveport, La., Radiology
62. Hayes, Arthur W., Greenfield, Mass., Diagnostic Roentgenology
63. Herrmann, Henry C., Louisville, Ky., Therapeutic Radiology
64. Hiebert, Peter E., Kansas City, Kans., Radiology
65. Hill, John M., Pittsburgh, Pa., Radiology
66. Hilt, Lawrence M., Grand Rapids, Mich., Radiology
67. Hobbs, Arthur A., Jr., Philadelphia, Pa., Radiology
68. Holtz, Harvey E., Dallas, Tex., Radiology

69. Howard, Corbett E., Goldsboro, N. C., Therapeutic Radiology
70. Howe, Martha E., New York, N. Y., Therapeutic Radiology
71. Hummon, Irvin F., Jr., Oak Park, Ill., Roentgenology
72. Hunter, Arthur F., New York, N. Y., Roentgenology
73. Jaffrey, George, Olean, N. Y., Radiology
74. Jelte, Safford A., Oakland, Cal., Radiology
75. Jenkins, I. Warner, Waco, Tex., Radiology
76. Jenkins, William F., Columbus, Ga., Roentgenology
77. Jensen, Herman H., Minneapolis, Minn., Radiology
78. Jimenez, Juan M. M., New York, N. Y., Radiology
79. Johnson, Roy W., Los Angeles, Cal., Radiology
80. Johnston, Wayne A., Dubuque, Ia., Roentgenology
81. Kelley, Charles H., Washington, D. C., Radiology
82. Kellogg, Douglas S., Ft. Sam Houston, Tex., Roentgenology
83. Kestel, John L., Waterloo, Ia., Diagnostic Roentgenology
84. Kirsh, Israel E., Salt Lake City, Utah, Radiology
85. Koenig, Edward C., Buffalo, N. Y., Roentgenology
86. Lake, William F., Atlanta, Ga., Roentgenology
87. Landham, Jackson W., Atlanta, Ga., Roentgenology
88. Larkin, John C., Jr., Berkeley, Cal., Radiology
89. Lavine, Sidney B., Trenton, N. J., Diagnostic Roentgenology
90. Lavner, Gerald, Haverhill, Mass., Diagnostic Roentgenology
91. Lawlah, John W., Chicago, Ill., Radiology
92. LeWald, Leon T., New York, N. Y., Radiology
93. Leader, Samuel A., Lexington, Ky., Diagnostic Roentgenology
94. Lerner, Henry H., Boston, Mass., Diagnostic Roentgenology
95. Levy, Abraham H., Brooklyn, N. Y., Radiology
96. Lewis, Elbert K., Chicago, Ill., Radiology
97. Lia, Bianca Rachel, Boston, Mass., Roentgenology
98. Liberson, Frank, Brooklyn, N. Y., Roentgenology
99. Logan, George E. C., Philadelphia, Pa., Radiology
100. McAlister, Lawrence S., Muskogee, Okla., Radiology
101. McCampbell, Herbert H., Knoxville, Tenn., Radiology
102. McCandless, Oliver H., Kansas City, Mo., Roentgenology
103. McCullough, Thomas L., Pittsburgh, Pa., Diagnostic Roentgenology
104. McGreer, John T., Dayton, O., Radiology
105. McKinney, G. C., Lake Charles, La., Roentgenology
106. McNattin, Robert F., Chicago, Ill., Therapeutic Radiology
107. Mackoy, Frank W., Milwaukee, Wis., Diagnostic Roentgenology
108. Magruder, Levin W., New Orleans, La., Radiology
109. Manley, Louis V., Northampton, Mass., Diagnostic Roentgenology.
110. Martineau, Lawrence A., Providence, R. I., Roentgenology
111. Mass, Max, Macon, Ga., Diagnostic Roentgenology
112. Maxfield, James R., Jr., Waco, Tex., Radiology
113. Meader, Robert P., Philadelphia, Pa., Radiology
114. Mokrohisky, Stephen M., Green Bay, Wis., Roentgenology
115. Moore, J. N., Ocala, Fla., Roentgenology
116. Morgan, Arthur E., Washington, Pa., Roentgenology
117. Morgan, Harold W., Mason City, Ia., Radiology
118. Morris, Clyde L., Peoria, Ill., Roentgenology
119. Myers, Karl J., Philippi, West Va., Radiology
120. Nash, Newman C., Wichita, Kans., Radiology
121. Neely, John M., Lincoln, Neb., Radiology
122. Newton, John O., Cleveland, O., Roentgenology
123. Norton, Richard C., Battle Creek, Mich., Diagnostic Roentgenology
124. Palmer, James P., Bronx, N. Y., Radiology
125. Payne, Andrew K., Jackson, Mich., Radiology
126. Pearlstein, Frank, West New York, N. J., Diagnostic Roentgenology
127. Pedersen, Nicholas S., San Francisco, Cal,. Roentgenology
128. Phillips, Allan B., Des Moines, Ia., Radiology
129. Piatt, Arnold B., Tulsa, Okla., Roentgenology
130. Plaut, H. F., Mansfield, O., Radiology
131. Pollack, Simon, St. Louis. Mo., Roentgenology
132. Popma, Alfred M., Boise, Idaho, Radiology
133. Poppel, Maxwell H., New York, N. Y., Roentgenology
134. Prince, Norman C., Amarillo, Tex., Roentgenology
135. Reed, Harold R., Washington, D. C., Radiology
136. Reid, Edward K., Rome, N. Y., Diagnostic Roentgenology
137. Rhinehart, B. A., Little Rock, Ark., Roentgenology
138. Richardson, Maurice L., Freeport, Ill., Roentgenology
139. Roberto, Romeo, Yonkers, N. Y., Diagnostic Roentgenology
140. Roberts, Edward W., Chicago, Ill., Roentgenology
141. Robilotti, James G., New York, N. Y., Diagnostic Roentgenology
142. Rodes, Charles B., Butte, Mont., Diagnostic Roentgenology
143. Rogers, Frank T., Ann Arbor, Mich., Radiology
144. Rubin, J. S., Jamaica, N. Y., Diagnostic Roentgenology
145. Sackett, George L., Cleveland, O., Radiology
146. Scott, Harry A., Lincoln, Neb., Diagnostic Roentgenology
147. Senturia, Hyman R., Chicago, Ill., Radiology
148. Sherman, Cyril F., Wichita, Kans., Roentgenology
149. Sickel, Emanuel M., Lakewood, N. J., Diagnostic Roentgenology
150. Silverstone, Sidney M., New York, N. Y., Therapeutic Radiology
151. Singer, Bella, Elizabeth, N. J., Diagnostic Roentgenology
152. Sirca, Dionisie M., Springfield, Ill., Roentgenology
153. Sisk, J. Newton, Madison, Wis., Radiology
154. Smith, Barney B., Buffalo, N. Y., Roentgenology
155. Smith, Earl D., Utica, N. Y. Radiology
156. Smith, Ernest E., New York, N. Y., Radiology
157. Sokow, Theodore, Kenosha, Wis., Radiology
158. Solomon, Bennet, Springfield, Mass., Diagnostic Roentgenology
159. Southworth, J. D., Rutland, Vt., Therapeutic Radiology
160. Spencer, Earle W., Saskatoon, Sask., Radiology
161. Stacy, Archie J., Tupelo, Miss., Diagnostic Roentgenology
162. Sternbergh, W. C. A., Clifton Springs, N. Y., Radiology
163. Stewart, Calvin B., Atlanta, Ga., Therapeutic Radiology
164. Szymanski, John J., Passaic, N. J., Radiology
165. Talley, Louis F., Marshalltown, Ia., Roentgenology
166. Thompson, William G., Holden, Mo., Radiology
167. Truog, Clarence P., Minneapolis, Minn., Roentgenology
168. Tyner, Furman H., Port Arthur, Tex., Radiology
169. Vaughn, Robert J., Annapolis, Md., Roentgenology
170. Wallace, William S., Durham, N. C., Radiology
171. Ward, Leo J., Elizabeth, N. J., Diagnostic Roentgenology
172. Warden, Ralph H., Chicago, Ill., Roentgenology
173. Webb, Harold H., Ottumwa, Ia., Radiology
174. Wehr, William H., Buffalo, N. Y., Therapeutic Radiology
175. Weinstein, Samuel, Brooklyn, N. Y., Roentgenology
176. Weitz, Harry L., Traverse City, Mich., Roentgenology
177. Williams, Edwin G. C., Danville, Ill., Therapeutic Radiology
178. Willis, Augusta Elizabeth, Orangeburg, S. C., Roentgenology
179. Wilson, Angus K., Norfolk, Va., Radiology
180. Wilson, Russell F., Beloit, Wis., Radiology
181. Worth, Harry M., Victoria, B. C., Radiology
182. Wright, Hobart H., Ann Arbor, Mich., Radiology
183. Zielinski, John B., Fall River, Mass., Radiology

RADIOACTIVE STANDARDS*

A series of radioactive standards are being prepared under the direction of the Committee on Standards of Radioactivity of the National Research Council. These standards will be deposited at the National Bureau of Standards in Washington, D. C.

* This work is being supported in part by a generous grant from the American Philosophical Society to the Massachusetts Institute of Technology.

to be issued as working standards to investigators who may desire them.

The standards under preparation at present are:

(1) Radium Standards

(a) 100 cc. solutions sealed in 200 cc. Pyrex flasks containing 10^{-9} and 10^{-11} grams of radium to be used as emanation standards either directly or by subdilution.

(b) 5 cc. solutions sealed in Pyrex ampoules containing 0.1, 0.2, 0.5, 1.0, 2.0, 5.0, 10, 20, 50 and 100 micrograms of radium to be used as gamma-ray standards. If desired, these may be obtained in sets of 13 with two each of the 0.2, 2, and 20 microgram standards.

(2) Thorium Standards

Sealed ampoules containing sublimed $ThCl_4$. These may be used in preparing standard thorium solutions.

Directions for use will be furnished with the standards.

(3) Standard Rock Samples

The following rocks, ground to pass 40-mesh screen and be retained on 100-mesh screen are available in 100 gram samples.

Quartzite (Virginia)
Triassic diabase (Virginia)
Milford granite (Massachusetts)
Chelmsford granite (Massachusetts)
Gabbro-diorite (Massachusetts)
Columbia River Basalt (Idaho)
Bearea sandstone (Ohio)
Dunite (North Carolina)
Carthage granite (Missouri)
Carthage limestone (Missouri)
Deccan Trap (India)
Kimberlite (South Africa)

These samples of rock will be analyzed for radium and thorium content and are intended for use as working standards to check methods used in extraction of radon and thoron from rock samples. They may be used for direct fusion in the electric furnace or for carbonate fusion.

All of the above samples will be analyzed at a number of laboratories equipped to make such measurements and ultimately certificates will be issued by the National Bureau of Standards. This work is in progress but will require considerable time for its completion so that final figures are available only for a part of the samples at the present time.

Accurate knowledge of the radioactive content of the materials of the earth's crust is of primary importance in many phases of geology, geophysics and cosmology. Reliable radioactive standards are also essential in studies of radium and thorium poisoning and in biological and medical investigations using the technique of radioactive indicators, or internal artificial radioactivity therapy. For the latter purposes calibrated standard sources of β-rays will be made available.

It is hoped that the standards which have been prepared by the Committee will provide all workers in these fields with a common basis for comparison of measurements and also improve the accuracy of all measurements of this type. It is likely that they will have other applications and the Committee would appreciate hearing from interested persons who may desire similar standards for their work. The Committee is also glad to cooperate as far as possible in aiding investigators to use these standards to the best advantage and welcomes specific inquiries regarding their use. It is urged that any suggestions regarding other desirable radioactive standards, not at present available, be submitted promptly to the Committee. In particular, it will facilitate the work of the Committee if those laboratories and individuals which can make use of these standards advise the Committee of their probable requirements.

Communications may be addressed to the Chairman, Professor Robley D. Evans, Department of Physics, Massachusetts In-

stitute of Technology, Cambridge, Massachusetts.

L. F. Curtiss
Clark Goodman
Alois F. Kovarik
S. C. Lind
C. S. Piggot
Robley D. Evans

CLINICAL CONFERENCE OF MID-WESTERN RADIOLOGISTS

The Fourth Annual Clinical Conference of Mid-Western Radiologists was held at the Brown Hotel, Louisville, Kentucky, on February 9 and 10, 1940. Presentations on many subjects of interest to radiologists were made by some of the leading physicians of Kentucky.

At the dinner meeting Dr. William Allen Pusey, of Chicago, Illinois, spoke on the subject "Reminiscences of my Early Experiences with the Roentgen Ray." He presented his office record and case history of the first patient he had ever treated with the roentgen ray, this being done early in the year 1900. He spoke of his therapeutic experiences with the roentgen ray in lesions of many types, many of which were undoubtedly so treated for the first time.

Dr. Irvin Abell, of Louisville, Kentucky, then spoke on the subject "The Present United States Public Health Program." In prefacing his major presentation he paid a glowing tribute to the radiologist. He spoke of his great importance in the general field of medicine and of the many contributions to medical progress that had been made by radiologists during the last forty years. He also commended radiologists in general for the work that they were doing in preserving their specialty and for their defense against the attempted inroads of so-called Social Legislation. He then gave a remarkable summary of the legislative activities of our Government in the field of medicine during the past six years, together with a summary of the legislation now before the Congress.

Dr. C. A. Good, of the Mayo Clinic, in behalf of Dr. B. R. Kirklin, invited the Conference to meet in Rochester, Minnesota, next year. The invitation was accepted by the Conference.

J. C. Bell

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers as space permits.

CHRONIC DISEASES OF THE ABDOMEN: A Diagnostic System. By C. Jennings Marshall, M.S., M.D. (Lond.), F.R.C.S. (Eng.), Surgeon, Charing Cross Hospital and Victoria Hospital for Children; Surgical Consultant, L.C.C., and Bromley District Hospital; Examiner in Surgery, London University and Victoria University, Manchester. Cloth. Price, $6.00. Pp. 247, with 131 illustrations. Boston: Little, Brown & Co., 1939.

As the author states in the preface, this volume is an endeavor to present in concise form for the use of the general practitioner the chronic conditions which affect the abdomen.

In Part I, he emphasizes that the abdomen cannot be considered apart from the rest of the body and in the notes on the history taking and physical examination it is pointed out that one must always think of the patient as a whole. His suggested physical examination is complete, although his avowed aim is determining abdominal troubles. A section on Special Investigations includes roentgenography, instrumental examinations such as proctoscopy, cystoscopy, gastroscopy, etc., and laboratory examinations such as urine, feces, blood, renal function tests, etc.

Part II deals with differential diagnosis and here the author takes up pain, and its significance, as it may arise from various abdominal systems and organs or is made manifest as referred from other anatomic sites such as the spine or chest. Intra- and extraperitoneal conditions are discussed and differentiated, and backache is taken up in detail. Finally, there is a section on "Other Symptoms" which covers hematemesis, jaundice, loss of weight, diarrhea, hematuria and vomiting.

The author has shown excellent balance throughout and always gives due emphasis to various symptoms. The limitations of different procedures are recognized and this is nowhere more evident than in the section on roentgenography which is perforce very abbreviated. Roentgenograms "are indispensable in many cases, in others of great help, but wherever they are employed merely as a short cut to avoid thinking, they are a menace."

Appropriate diagrams and roentgenograms illustrate the book. Nowhere does one get the impression of "padding." For the roentgenologist who wishes a good yet brief treatise on the "chronic abdomen," this book can be recommended.

H. D. KERR

MALADIES OSSEUSES: MALADIE DE RECKLINGHAUSEN, MALADIE DE PAGET, LIPOIDOSES OSSEUSES, MYELOMES MULTIPLES. Par Dr. I. Snapper, Professeur à l'Université d'Amsterdam. Traduction du Professeur dr. F. de Witte (Gand) et le dr. G. Coryn (Bruxelles). Cloth. Price, 330 fr. Pp. 192, with 22 plates. Paris: Masson et Cie, 1938.

In this volume, chronic cystic osteitis or von Recklinghausen's disease of bone, osteitis deformans or Paget's disease, xanthomatosis, Gaucher's disease and multiple myeloma are described in detail. Almost one-half (84 pages), is devoted to von Recklinghausen's disease.

In the first chapter, a review of the literature of chronic cystic osteitis is followed by a discussion of the relationship between it and the pathological changes found in the parathyroid glands, a description of the microscopical changes found in hyperplasia and adenoma of the parathyroids and a review of the criteria on which to base a differential diagnosis of these pathological changes. The roentgenological characteristics, the pathological variations in calcium and phosphorus metabolism and the microscopical skeletal changes are described in ample fashion. Case reports of patients who were operated upon by Snapper for removal of adenomata of the parathyroids are included. In these patients, recalcification of the skeleton occurred after operation. In the second and third chapters, the question of hypertrophy of the parathyroids caused by primary disturbances of calcium metabolism, and the effect of removal of normal parathyroids in ankylosing arthritis, scleroderma and other diseases are discussed.

The description of Paget's disease is covered in a chapter of 30 pages. While the symptomatology, etiology, biochemical changes,

roentgenographic characteristics and other features are fully described, the omission of any reference to osteoporosis circumscripta is worthy of mention. The remaining chapters are devoted to xanthomatosis, Gaucher's disease and multiple myeloma. Throughout emphasis is placed on the differential diagnosis of these various conditions and von Recklinghausen's disease.

The book is illustrated with 22 plates reproducing roentgenograms, microscopical and gross specimens and clinical photographs of the diseases described. The illustrations are clear and are excellent from every point of view. The bibliography following the chapters on von Recklinghausen's disease is voluminous but the number of references for the other diseases is, in comparison, small. However as the volume is actually a monograph on chronic cystic osteitis and its differential diagnosis, this is not a serious defect. It can be recommended as a concise reference book for those interested in the diagnosis and treatment of these diseases of bones.

R. S. Bromer

End-Results in the Treatment of Gastric Cancer: An Analytical Study and Statistical Survey of Sixty Years of Surgical Treatment. By Edward M. Livingston, B.Sc., M.D., Associate Visiting Surgeon, Bellevue Hospital, New York; Assistant Clinical Professor of Surgery, New York University College of Medicine, and George T. Pack, B.Sc., M.D., F.A.C.S., Attending Surgeon, Memorial Hospital, New York City; Assistant Professor of Clinical Surgery, The School of Medicine, Yale University, New Haven and Cornell University Medical College, New York City. With a Foreword by Bowman C. Crowell, M.D., Associate Director, American College of Surgeons. Cloth. Price, $3.00. Pp. 179, with numerous charts. New York: Paul B. Hoeber, Inc., 1939.

This small volume, prepared as a single chapter in a comprehensive treatise on the treatment of cancer and allied diseases soon to be published, contains a careful analysis of the results produced by surgery in cancer of the stomach, beginning with the first successful resection performed by Billroth almost sixty years ago. Although one-third to one-fourth of all cancers occur in the stomach, the absolute curability of the disease at the present time amounts to less than 5 per cent. An attempt is made to offset this depressing figure by repeatedly emphasizing the observation that of the 30,000 to 50,000 cases developing in the United States each year, approximately 10,000 are resectable. Since this incidence equals that of cancer of the breast, and is much higher than that of many other forms of cancer, the authors believe that resectable cancer of the stomach should be considered as a separate entity in which the prognosis is no worse than it is in many other forms of malignant disease. From a study of 14,000 operations collected from the better surgical clinics of the world, and tabulated in the back of the book the conclusion is reached that good surgery carried out by properly trained teams should permanently cure at least 20 per cent of the resectable group. Such reasoning will serve as a stimulus to those who undertake radical operative procedures, but it offers little solace to the 28,000 to 48,000 patients who obtain little relief and die each year.

An elaborate supplement contains thirty charts and tables beautifully prepared as a graphic presentation of the points made in the text. Because of the technical excellence of this material lantern slides can easily be prepared from it for teaching purposes. The bibliography includes 358 references and is very complete. In another supplement, a study of statistics for this country, compiled over a fifteen year period, shows 244,357 deaths from war, 441,912 fatalities from traffic injuries, and an estimated death rate of 600,000 from cancer of the stomach. These figures are graphically depicted in poster form on the fly-leaves at the front and back of the book.

Charles L. Martin

The Construction of Vulcanite Applicators for Applying Radium to Lesions of the Buccal Cavity, Lips, Orbit and Antrum. By Desmond Greer Walker, M.A., M. Dent. Sc., M.B., B.Ch., Assistant Dental Surgeon to the Royal Dental Hospital; Dental Registrar at the Middlesex Hospital; Chief Assistant to the Plastic Department, St. Bartholomew's Hospital. Foreword by W. Warwick James, O.B.E., F.R.C.S., L.D.S. Cloth. Price, 5/- Pp. 61, with 23 plates. London: John Murray, 1938.

This small sixty-one page book is prepared

by a dentist for the guidance of others in his profession who may be called upon to prepare surface radium applicators for the treatment of malignant tumors in the mouth and cavities of the bones of the face. The principle of using multiple small, heavily filtered radium sources fixed a short distance from the tumor surface is advocated and the applicators are carefully made so that they may be worn with comfort over considerable periods of time. The distance factor is provided by a thin layer of Columbia paste placed in a protective shield of lead and covered by a carefully fitted vulcanite shell made to accurately conform to the part treated. When the completed device is assembled it is, in many instances, held in place by bars attached by springs or straps to a head band. Numerous photographs included in a series of 23 plates illustrate the method adequately, and careful directions are given for the preparation of the materials used. For those who elect to utilize this elaborate form of radium therapy which has much to commend it, especially in the care of superficial lesions overlying bony surfaces, the book should prove most valuable.

Charles L. Martin

Sobre a radiologia do canal optico. By H. C. P. Marback, Bahia, Brazil. Pp. 89, with 45 plates. Bahia: Imprensa Regina, 1939.

This monograph, written as a competitive thesis, reviews the embryologic development, anatomic structure and anthropomorphic determination of the positions of the optic canals. It mentions and comments on the various techniques (some 30 in all) employed for making roentgenograms of these structures. The author then presents his own improved technique and recommends it for general use.

In this method a cage-like device is attached to the roentgen tube to fit over the patient's head and hold a small film perpendicular to the primary pencil of rays. In operation the patient is seated with the head steadied on a chin support, with the Virchow plane (plane of Reid's line) corresponding to the horizontal plane. The device is placed over the head, and the tube "must be moved in such manner as to annul angles alpha and beta" of the optic canals. These angles are achieved by directing the tube downward for the angle alpha (25 degrees mean) and obliquely for the angle beta (37 degrees). With the tube set at these angles the device must be lined up with the patient's head, so that the primary rays strike the skull at Hartmann's point, to pass through the canal and emerge at Brunetti's point (center of the lower and outer quadrant of the orbit) to expose the film.

Marback's technique is not adaptable to routine practice and does not fill the requirements for a practical method. Too much is left to the judgment of the technician. A number of exposures are recommended by Marback for each canal. Thus true contours apparently are not consistently obtained. The method makes the projection of the canals a special procedure, several pieces of special apparatus being required. It does not allow for symmetrical projection of the canals, a requirement which is essential for their comparison, for it is not enough to examine only one canal in any given case. Stereoscopic projections can scarcely be made with the technique.

Raymond L. Pfeiffer

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 105 Rutledge Ave., Charleston, S. C.

SIMPLE METHOD FOR ROENTGENOGRAPHY OF THE OPTIC CANAL

By M. MORRIS WITTENBERG
BROOKLYN, NEW YORK

THE optic foramina can be perfectly demonstrated by the use of the Bullitt mastoid apparatus.

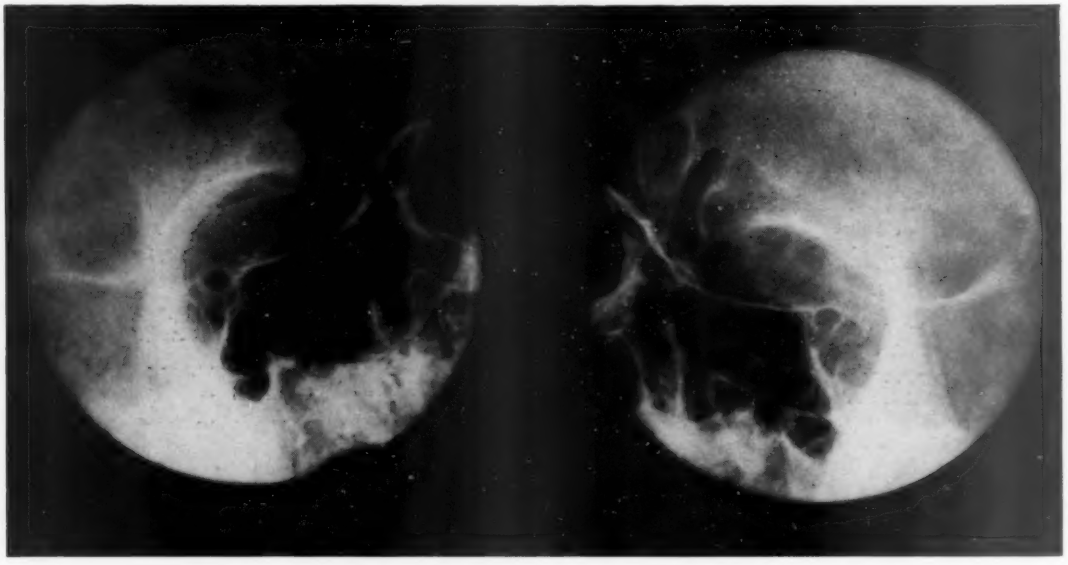


Fig. 1. Roentgenogram showing both canals and some of the accessory sinuses.

With this apparatus identical comparative studies can be made of both sides and duplicated at any future date (Fig. 1). The procedure is simplified considerably since the orbit to be examined is constantly visible throughout the study and the head is perfectly immobilized. There is no necessity for measuring devices or for focusing the tube. This mastoid apparatus is already in use in most hospitals and private offices so that no additional apparatus is necessary.

The patient is placed in the supine position with the head rotated obliquely so that the corresponding orbit touches the cellulose sheet. The center of the orbit is placed at the crosslines and the head is immobilized in the manner provided by the apparatus (Fig. 2). The patient may be studied in

Fig. 2. Apparatus with patient in position. Notice the relationship of the orbit to the crosslines.

prone position with equally satisfactory results and stereoscopic studies may be obtained by utilizing the shift. The posterior ethmoidal and sphenoidal sinuses are visualized as with the Rhese position and again permits an exact comparison and duplication of both sides.

The exposure necessary and the masking device used are the same as with mastoid studies (Fig. 3) except when the examination is made in the prone position. The sides are then reversed and a special blocking out mask is necessary.

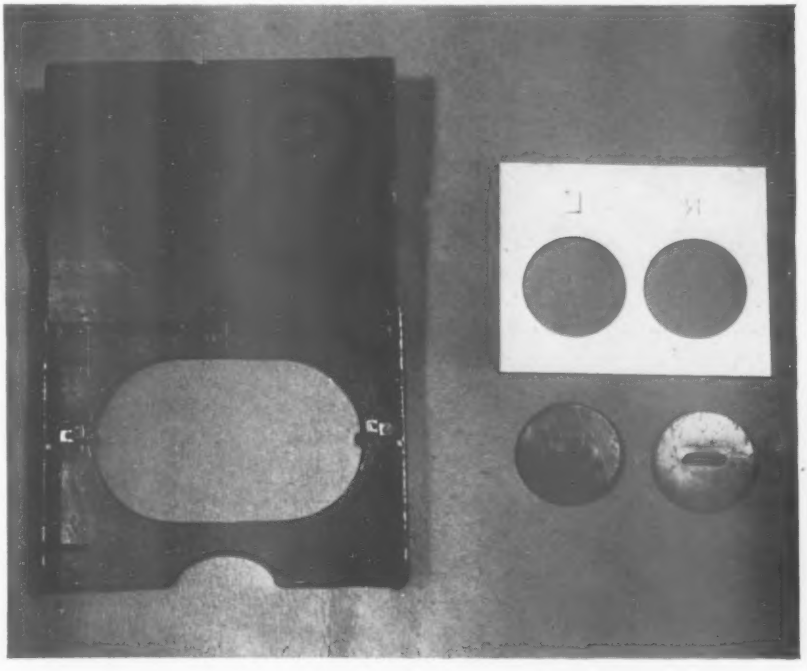


Fig. 3. Bullitt masking device and the one used when employing the prone position.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

Jones, William A. Further observations regarding familial multilocular cystic disease of the jaws. *Brit. J. Radiol.*, April, 1938, *11*, 227–240.

Author's Summary: Further observations regarding a peculiar condition occurring in three members of the same family are given. These cases were first described by the author at the meeting of the Radiological Society of North America at St. Louis, Dec. 1, 1931, and published in the *American Journal of Cancer*, April, 1933. The condition is one of multiple dentigerous cysts occurring universally throughout both the upper and lower jaws, and accompanied by a chronic hyperplastic disorder of the cervical lymph glands of the submaxillary regions.

The condition presents itself toward the end of the second year of life and is accompanied by bilateral painless swelling of both upper and lower jaws.

Roentgenograms and photographs of these children made in 1931, and again in 1937, are shown, and the course of the disease is discussed. A fourth member of the family has escaped and presents normal dental development. A fifth member of the family (sixteen months old) already presents a suspicious bulge at the angle of the left side of the lower jaw. There is no evidence of a like condition in a previous generation of either side of the family.

Through the kindness of Dr. P. J. Thomas, of Savannah, Ga., the author has at his disposal the description, with roentgenograms and photographs, of a similar case in a boy now eleven years of age. In this case the multilocular cysts of both jaws are also observed. There is, however, in this patient, no evidence of lymphatic glandular involvement. Dr. Thomas has produced a genetic chart showing the descent of the anomaly through five generations of the family of this patient.—*S. G. Henderson.*

Blady, John V., and Hocker, Alfred F. Sialography, its technique and application in the roentgen study of neoplasms of the parotid gland. *Surg., Gynec. & Obst.*, Dec., 1938, *67*, 777–787.

The technique, indications and contraindications of sialography are discussed. Inasmuch as no harmful effects from its use have been noted, it appears that its use should be more generally accepted. The authors have found it of value in the determination of the relationship of the normal duct system and gland tissue to adjacent structures, for the demonstration of salivary fistulae, duct stones, foreign bodies or strictures, in diagnosing chronic recurring inflammation, and to determine the nature of a growth as to whether it is encapsulated, infiltrating or is a mass extrinsic to the gland and ducts proper. The irregular filling defects in the gland structure and the filling defects in the finer ramifications of the duct system produced by an infiltrating tumor are quite characteristic. The knowledge gained from the sialogram will help determine the operative plan more accurately prior to operation and herein lies its greatest value.—*P. C. Swenson.*

Swinton, Neil W., and Warren, Shields. Salivary gland tumors. *Surg., Gynec. & Obst.*, Oct., 1938, *67*, 424–435.

Eighty-one tumors of the salivary glands are reported. The concepts of origin of these tumors are thoroughly discussed. The authors favor the concept that the tumors arise from misplacement of cells during the rather complex embryonic development of the face. They present 51 mixed tumors with clinical data. Their variable histological picture is stressed, as well as some of the less common benign tumors. Of the mixed tumors operated upon at the Lahey Clinic 4.3 per cent recurred. Careful complete excision of the salivary gland tumor without rupture of the capsule or spilling of its contents must be done if recurrence is to be prevented. This may involve sacrificing the facial nerve. Although the results of radiotherapy have not been encouraging, the authors feel that with further experience the results may be improved. Combined radical excision and radiotherapy should be continued in all malignant tumors until it is definitely proved

that radiotherapy is of no value. Three of 9 malignant tumor cases are alive and well two and four years after operation. The diagnosis of malignancy is based on the finding of combined cellular anaplasia or metaplasia, invasive properties, and evidence of rapid growth in whatever tissue these changes might make their appearance. Invasive growth alone, although ordinarily a helpful diagnostic point in the case of malignant tumors, is valueless in the group of salivary gland tumors.—*P. C. Swenson.*

NECK AND CHEST

Ray, Bronson S., Tumors at the apex of the chest. *Surg., Gynec. & Obst.*, Nov., 1938, *67*, 577–599.

Five cases of apex tumors are added to the literature, all of different histologic structure but of similar clinical manifestations as in the cases described originally by Pancoast and others. The literature is reviewed. The neurological aspects of the cases are emphasized, particularly the surface pain and its segmental distribution, the sensory changes, sympathetic irritation, cord compression, etc. There is very little if any help from roentgen or radium therapy. Treatment must be directed toward the surgical relief of pain wherever possible, and this makes early diagnosis important.—*P. C. Swenson.*

Friedman, Townsend B., and Molony, Clement J. Rôle of allergy in atelectasis in children. *Am. J. Dis. Child.*, August, 1939, *58*, 237–249.

The authors report 6 cases of nontraumatic, nonpostoperative atelectasis and 1 case of postoperative atelectasis occurring in allergic children. They conclude that the processes in bronchial asthma which may produce bronchial occlusion are bronchospasm, thickening of the bronchial walls, secretion of thick mucus and paradoxic collapse of the bronchi during expiration. All evidence seems to point to bronchial occlusion as the principal cause of atelectasis. They suggest the possibility of recurrent atelectasis as a precursor of bronchiectasis.

The paper is profusely illustrated with reproductions of the roentgenographic changes in the lungs in the cases of their series. They regard the treatment as twofold. One is concerned first with the immediate relief of the patient and second with methods of preventing recurrence. Treatment is directed at removing the mucous plug from the bronchus. The ordinary indirect methods are postural drainage and the use of expectorants and drugs which increase the bronchial secretion or render it more liquid. Epinephrine hydrochloride given subcutaneously, while it makes the patient more comfortable, does not serve to release the tenacious, thick mucoid plugs. If the condition persists, they believe that immediate bronchoscopic aspiration is the method of choice, and the aspiration may have to be repeated until the pulmonary tree is free of obstruction.

In order to prevent recurrence a thorough allergic study of the cause of the asthmatic attacks should be made and by preventing the seizures, recurrences of the atelectasis should be eliminated.—*R. S. Bromer.*

Kautzky, A. Grenzen bronchographischer Diagnostik der beginnenden Bronchiektasienbildung. (Limits of bronchographic diagnosis in early bronchiectasis.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Feb., 1938, *57*, 168–176.

The fully developed case of bronchiectasis with definite dilatation of the bronchi offers no difficulty in diagnosis. The border line cases are difficult. The bronchi normally become narrower as one approaches the periphery. When this progressive narrowing stops, a dilatation is present. Occasionally, however, there is widening of a bronchus without a true ectasia being present, and, on the other hand, differences sometimes occur between the bronchographic and the autopsy findings. The indirect signs of bronchiectasis are a failure of the alveoli to fill with lipiodol, a tortuosity of the bronchi, and third a failure of the lipiodol to show a peripheral motion during inspiration. This latter sign occurs in all parenchymal diseases and indicates a defective areation of that portion of the lung. These three signs may be present before a definite ectasia is demonstrable and may lead to an earlier diagnosis. Repeated observations over a long period are desirable.—*W. A. Evans, Jr.*

Boyd, Linn J., and McGavack, Thomas H. Aneurysm of the pulmonary artery. *Am. Heart J.*, Nov., 1939, *18*, 572–578.

Aneurysm of the pulmonary artery is rare. About 139 cases had been reported up to 1936. By definition the term is limited to those

dilatations which are the result of, or associated with, actual damage to one or more coats of the vessel. The sex distribution is about equal and the mean age 37.7 years with 34 per cent of the cases under thirty years of age. The trunk of the artery is most commonly affected and the saccular dilatation is a little more common than the fusiform type. Right and left branches are involved equally often. In the etiology, trauma seemed to be a factor in 3 cases, congenital anomalies were significant in about one-half, and arteriosclerosis was regarded as a provocative in 23 per cent. Syphilis was regarded as responsible for all cases by Warthin, but the figure is rare, probably 30 per cent and that may be too high. In a few cases, septic or rheumatic endocarditis appeared of etiologic importance.

The common symptoms are dyspnea, cyanosis, pain, palpitation and cough. On physical examination there is enlargement of the mediastinum toward the left at the second and third costal cartilages and in 2 cases there was erosion and necrosis of these cartilages. A harsh systolic murmur is usually heard in this area. Right-sided cardiac enlargement is almost constantly present at autopsy, but is often missed during life.

Roentgenologically there is observed to be a prominence in the region of the pulmonary conus which pulsates if a thrombus is not present and gives a "seesaw" movement to the left side of the silhouette. In the right anterior oblique view there is encroachment on the retrosternal space and there may be a knob-like projection into the posterior mediastinum beneath the aortic arch.

In the differential diagnosis, aortic aneurysm, patency of the ductus arteriosus, idiopathic dilatation of the pulmonary artery, and various congenital malformations must be considered.

The authors report 2 cases, one a syphilitic Portuguese, aged forty-two, with an aneurysm localized in the left main branch of the pulmonary artery by the method of Robb and Steinberg, and also at operation performed under the mistaken diagnosis of carcinoma of the lung. The second case was a male syphilitic, aged sixty-one, who exhibited roentgenoscopically a dilatation of the pulmonary conus and a superimposed semilunar shadow the size of a peach. In both cases the aneurysms were regarded as syphilitic in character.—*W. A. Evans, Jr.*

Teschendorf, Werner. Röntgenologische Herzfunktionsprüfung. (Roentgenologic tests of cardiac function.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, Sept., 1939, *60*, 214–235.

There are four roentgen methods of testing circulatory function: (1) kymographic studies of the heart before and after exercise; (2) the method of de Abreu involving the correlation of changes in the surface area of the heart with cardiac volume and body weight; (3) measurements of the cardiac volume before and after exercise; (4) the Valsalva test.

In regard to the first method, the normal heart exhibits a diminution in size following exertion with an increase in the amplitude of the pulsation. In cardiac insufficiency there is an increase in the size of the heart and a diminution in amplitude following over-exertion. Care must be taken that the exposures are made in the same phase of respiration to insure a constant position of the diaphragm leaves.

The method of de Abreu is concerned with the relation of the volume change in the cardiac cycle measured kymographically to the cardiac volume and the body weight. For these measurements the author has used an egg-shaped vector kymograph and the values are introduced into the formula $e.v. = D.P.t.$ where e is the systolic retraction of the cardiac border, v the heart volume, D the pulse volume, P the blood pressure and t the work time. In the formula $e.v./P$ where P is the body weight the value should be about 4 for men and 3.6 for women.

The heart volume is best measured by the method of Lysholm with simultaneous postero-anterior and lateral exposures and application of the Rohrer-Kahlstorf formula. Measurements before and after exercise show a diminution of volume in the normal heart while there is an increased volume in the heart of impaired efficiency.

The author highly recommends the quantitative Valsalva test carried out by the method of Bürger before and after exertion. Some modifications are necessary for gravely ill patients and the contraindications are aortic and cardiac aneurysm, advanced coronary artery disease, heart block, and severe cardiac insufficiency. The normal heart decreases in size on both sides during the test. After exercise the decrease is less marked on one or both sides. The left side does not partake of the

decreased size in hypertrophy of the left ventricle. The failure of the right side to diminish is uncommon and usually accompanies congestion of the lesser circulation. If the heart does not decrease in size at all it is a sign of severe insufficiency and carries a poor prognosis. A fifth possibility is that there is a greater dimunition in size after exercise and this occurs with flabby atonic hearts and a poor prognosis.—*W. A. Evans, Jr.*

ASH, RACHEL, WOLMAN, IRVING J., and BROMER, R. S. Diagnosis of congenital cardiac defects in infancy. *Am. J. Dis. Child.*, July, 1939, *58*, 8–28.

This report consists of an analysis of 32 cases of cardiac anomalies which were verified at autopsy. Of the 32 cases, a correct diagnosis was made or suggested in only 14. An attempt is made to evaluate the diagnostic features associated with congenital heart lesions in infants. The following symptoms and physical signs are discussed in detail: cyanosis, polycythemia, thrills, murmurs. There is also a discussion of the electrocardiographic and roentgen findings. The authors comment on the clinical features of the more usual cardiac defects, interventricular septal defect, interatrial septal defect, patency of the ductus arteriosus, pulmonic stenosis, the tetralogy of Fallot, complete transposition of the great vessels, defective development of the right ventricle associated with atresia or hypoplasia of the tricuspid valve, and anomalies of the coronary vessels.

The roentgenologist can determine whether the individual chambers of the heart are large or small and whether abnormal pulsations or other vascular changes are in evidence, but he should not be expected to indentify intracardiac lesions. As a result of the increase in the cardiac shadow to the right of the sternum in the anteroposterior view, an enlarged right atrium gives rise to the appearance of a globular heart situated in the midposition. In the noncyanotic infant the most frequent cause of such a globular heart is a defect of the interatrial septum, usually persistent patency of the foramen ovale. Enlargement of the conus of the right ventricle is always associated with some degree of fullness in the left median or pulmonic arc. Marked enlargement of the cardiac contour to the left not infrequently is due solely to hypertrophy of the right ventricle. This can be verified by noting anterior bulging of the cardiac shadow in the lateral or the oblique view, with no posterior cardiac enlargement. The position of the left apex fails to differentiate between enlargement of the left and enlargement of the right ventricle. Aneurysmal enlargement of the pulmonary artery from any cause reveals itself as a rounded prominence in the pulmonic region. In the authors' series it was associated with (*a*) patency of the ductus arteriosus, (*b*) stenosis of the pulmonic valve and (*c*) dilatation of the pulmonic valve, interventricular septal defect and dextroposition of the aorta (Eisenmenger's complex).

The pulmonary artery distal to a malformed, contracted valvular ring is often dilated and appears in the roentgenogram as an enlarged pulmonary arc. But if, as happens more frequently, there occurs developmental hypoplasia of the infundibular region of the right ventricle and pulmonary artery, concavity or sharp angulation in the region of the pulmonic arc will be seen.

Atrophy of the left ventricle may manifest itself in lateral and oblique views as a flattening of the posterior outline of the heart with increase in the clear space between the heart and the chest wall. If the right ventricle is of normal size, hypertrophy of the left ventricle should be associated with diminution of the clear space posterior to the heart. However, if the heart consists largely of an enormous left ventricle, the right ventricle being merely a small appendage, the left may then be displaced anteriorly and no encroachment on the posterior clear space is shown but rather a seeming anterior enlargement simulating hypertrophy of the right ventricle.

Either pulmonary or aortic atresia and transposition of the great vessels may present a narrowed mediastinal shadow on roentgenoscopic examination. In the oblique position, this shadow should remain unchanged in the presence of atresia of either of the great vessels but should broaden out in the presence of transposed vessels.

A narrow mediastinal shadow associated with transposed vessels in the anteroposterior roentgenogram occurred in only 25 per cent of the cases of the series reported. More frequently the tremendously enlarged right auricular appendage projecting upward into the mediastinum caused a widening of the mediastinal shadow on the right side. When in

association with transposition of the great vessels the left ventricle became hypertrophied, there seemed to be a special tendency toward dilation of the right atrium and auricula.

In interventricular septal defect, the roentgen contour is unchanged, or some degree of hypertrophy of both the right and left ventricle may be noted in the lateral and oblique view. Interatrial septal defect is shown in the anteroposterior view as definite enlargement to the right of the sternum due to enlargement of the right atrium. The characteristic outline is globular. The hilar shadows are increased. Hypertrophy of the right ventricle may be noted in lateral and oblique views. If in a noncyanotic infant presenting a murmur of the type associated with an interventricular septal defect, the heart shows definite enlargement of the right atrium together with marked enlargement of the right and perhaps also of the left ventricle as observed in lateral and oblique views, there probably exists both an interventricular septal defect and an interatrial defect.

In patency of the ductus arteriosus, the roentgenogram shows most frequently enlargement of the right ventricle, with some prominence of the pulmonic arc and increased hilar shadows. Under the roentgenoscope increased pulsations are visible in the pulmonary artery and the hilar regions.

In pulmonic stenosis the roentgenoscopic examination should show the hilar densities as small in comparison with the prominent pulsating shadows typically associated with patency of the ductus arteriosus.

In the tetralogy of Fallot, the roentgen appearance varies with the site of the lesion. If there exists the customary developmental hypoplasia of the conus of the right ventricle and of the pulmonary artery, a concavity or a sharp angulation may be present in the region of the pulmonic arc. On the other hand, stenosis of the cusps with dilatation of the vessel beyond the site of the stenosis may be associated with an enlarged pulmonic arc. In addition the mediastinal shadow is usually enlarged to the right, and lateral and oblique views will demonstrate hypertrophy of the right ventricle. Not infrequently hypertrophy of the right ventricle is so pronounced that the lower pole of the cardiac apex is formed by the right ventricle, the left ventricle being displaced upward. In such cases the apex of the cardiac shadow may have a blunt, straight appearance which has been likened to the tip of a wooden shoe (coeur en sabot).

In transposition of the great vessels, the roentgenoscopic examination may be narrow in the anteroposterior view but should broaden out in the lateral and oblique views.

In defective development of the right ventricle associated with atresia or hypoplasia of the tricuspid valve, the diminutive size of the right ventricle is seen in the roentgenogram (*a*) in the anteroposterior view, as a concavity in the region of the pulmonic conus, and (*b*) in the left anterior oblique view, as absence of cardiac shadows anterior to the shadow of the aorta.—*R. M. Harvey.*

Giering, John F., and Charr, R. The heart in anthracosilicosis. *J. Am. M. Ass.*, Aug. 12, 1939, *113*, 574–576.

Congestive heart failure is a common occurrence in cases of far advanced anthracosilicosis. In a series of postmortem examinations made on anthracite miners, heart disease was found to be twice as common in the miners as in nonminers. In the present study only the miners without clinical evidences of congestive heart failure were studied. Twenty-five miners were studied. The ages of the miners varied from thirty-six to sixty-seven. The duration of their occupation varied from three to forty years. All the miners had either moderately or far advanced anthracosilicosis, as evidenced by either nodular or massive consolidation in the lungs revealed by roentgenographic examinations. Nine, in addition to anthracosilicosis, had either moderately or far advanced pulmonary tuberculosis.

The blood counts showed secondary polycythemia in most instances. The vital capacity was reduced in all cases in direct proportion to the extent of the anthracosilicosis. The venous pressure in most cases was within the normal limits. The velocity of the pulmonary and of the complete circulation was practically normal in all the cases. Roentgenographic examination of the heart showed no enlargement except in 2 cases. The aortic knob was prominent in most cases. The functional tests showed that practically all the miners studied had abnormal acceleration of the pulse rate after slight exertion. The electrocardiograms in the majority of instances showed myocardial damage.—*S. G. Henderson.*

ABDOMEN

Rivers, Andrew B. The importance of cancer as a cause of chronic dyspepsia. *J. Am. M. Ass.*, Sept. 23, 1939, *113*, 1188–1192.

The object of the study reported in this paper was to gather some information relative to the frequency with which cancer is found to be the cause of indigestion. The organ most frequently invaded by cancer responsible for dyspepsia was the stomach. Next in order of frequency was the pancreas. Carcinoma of the pancreas is much more frequently found in men than in women. This is true also of carcinoma of the esophagus. The author concludes that:

1. Dyspepsia is caused by cancer much more frequently in men than in women.
2. Cancer begins to become a relatively frequent cause of indigestion in both sexes at about the age of forty-five.
3. There is a pronounced increase in the incidence of cancer as the cause of indigestion in later life. In this series, of the men beyond the age of seventy years who had a primary complaint of indigestion 58 per cent were found to have malignant disease.
4. Men seem to progress into a definite dyspepsia-cancer age from five to ten years earlier in life than women. This age in men started in the late fifties and in women between the ages of sixty-five and seventy years.
5. After the age of forty-five symptoms of dyspepsia should be evaluated with meticulous care and this should include roentgenologic investigation.
6. An educational program having for its purpose the enlightenment of the public with regard to the hazards of nonprofessional treatment of dyspepsia after the age of forty-five, and particularly throughout the dyspeptic-cancer age period would produce some highly desirable results.—*S. G. Henderson.*

Carter, R. Franklin, and Collins, Harold L. Anomalies of the bile ducts; report of two cases with operations and autopsies. *Am. J. Dis. Child.*, July, 1939, *58*, 150–161.

The authors report 2 cases of anomalies of the biliary tract. In one there was cystic dilatation of the extrahepatic ducts with an unusual arrangement and number of ducts; in the other, stenosis at the papilla of Vater.

The symptoms of cystic dilatation of the bile ducts are variable, and the condition is seldom diagnosed preoperatively. The condition is usually found in a person about eighteen years of age who has had symptoms referable to the biliary tract, such as nausea, pain and intermittent attacks of jaundice. At times a tumor may be palpated in the right upper quadrant of the abdomen. The clinical picture of congenital atresia is that of complete obstruction of the biliary passages. Jaundice may be present at birth, but more often the child appears normal, becoming jaundiced three or four days later. This jaundice usually increases to the point where at three to four months the infant has a grayish-green color. The liver increases in size but the nutrition remains good. The condition must be differentiated from icterus neonatorum, syphilitic hepatitis, congenital hemolytic icterus, jaundice of sepsis and jaundice due to enlarged lymph nodes. When the diagnosis of cystic dilatation or atresia of the ducts has been made, an exploratory laparotomy is indicated.—*R. S. Bromer.*

Meves, F. Röntgenbefunde bei der chronischen Amöbenruhr. (Roentgen findings in chronic amebic dysentery.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, August, 1939, *60*, 175–180.

These studies were carried out at Tübingen from 1935 to 1937 by means of barium enema with films before and after expulsion of the barium and also with the air contrast study of the colon. All patients were carefully studied clinically, bacteriologically, and rectoscopically. A segmental colitis is common in chronic amebic dysentery. The affected parts of the colon appear atonic with an absence of the normal haustrations and with very fine toothline serrations of the wall. They are very sensitive to palpation. After evacuation the ulcerations of the mucosa may be seen. Deep penetrating ulcers were encountered very rarely in this group of cases. Where the bowel was very atonic the mucosal relief pattern was not obtained. As a rule films exposed immediately after defecation show emptying only of the sigmoid and rectum except in severe cases with acute symptoms, where complete emptying is a diagnostic sign. Sometimes hypophysin or prostigmin was useful for atony of the colon.

but the effect was by no means constant. In general, one may say that in no stage of amebic dysentery is there a characteristic roentgen appearance, and there is no special site of involvement. Clinical improvement is accompanied by a corresponding improvement in the roentgen appearance although thickening of the wall of the colon and irregularity of the mucous membrane may persist. The author encountered no cases of stricture such as have been described by Bassler between the sigmoid and the rectum. A recurrence of symptoms should not be carelessly attributed to an exacerbation of the amebic dysentery; a carcinoma was encountered in such a case.—*W. A. Evans, Jr.*

Rosser, Curtice, and Kerr, J. G. Cancer of the rectum in young persons. *J. Am. M. Ass.*, Sept. 23, 1939, *113*, 1192–1194.

Several writers since 1910 have called attention to an apparently increasing incidence of carcinoma, more particularly carcinoma of the large intestine, in young persons. The authors analyze a series of 100 consecutive cases of rectal cancer to determine the relative incidence of this disease in persons thirty-five years of age and under, the grade of malignancy present in younger persons as compared with that in more mature years and such other factors as might enter into the prognosis of the younger age group.

In this series 2 per cent were sixteen years of age, 7 per cent were under thirty and 17 per cent were under thirty-six. The average duration of symptoms for the younger age group was seven months. There was no evidence that the grade of malignancy present in this group was higher in young persons, but colloid cancer was found more frequently and squamous cell cancer less frequently. The younger group provided better surgical risks, and resection was attempted in a greater number than in older persons.

The authors conclude that since this younger group, in contradistinction to some other published reports, does not show a greater percentage of higher grades of carcinoma, it must be assumed that the earlier spread of the lesion, indicated by shorter than average duration of symptoms and early gross involvement of lymph nodes and neighboring structures, is due to a partial absence of defensive barriers to early spread of the lesions in young persons.—*S. G. Henderson.*

GENITOURINARY SYSTEM

Williams, E. Rohan. Urography in pregnancy. *Brit. J. Radiol.*, April, 1938, *11*, 202–210.

A review of various papers shows that in normal pregnancy the right kidney almost always shows pelvi-ureteric dilatation. Both sides are affected in one-half of the cases, but it is rare to find dilatation only on the left side. Various causes of physiological dilatation and adynamia are: (1) obstruction from the enlarging uterus; (2) hypertrophy of the lower ureter. In pregnant women without urinary infection a concentric hypertrophy occurs in the lower zone of the ureter; (3) hormonic influence. Something in the blood of pregnant women prevents the uterus from emptying itself until full term. While this uterine inhibition is present, so also is there a relative adynamia of the pelvi-ureteric musculature. Urographic changes in normal cases are: (1) normal excretion rate from kidney; (2) slow pelvi-ureteric clearance; (3) moderate dilatation with a column effect from pelvis to ureter.

The changes in pyelitis of pregnancy differ really only in degree from the physiological changes. Six to 8 per cent of pregnant women suffer "pyelitis." In moderate and severe types there may be seen a marked depression of excretory function of the kidneys and marked dilatation, especially of the minor calyces.

The roentgenological diagnosis of placenta praevia should be extremely accurate with the aid of cystography. The bladder is catheterized and emptied and 2 to 3 ounces of sodium iodide are instilled. Roentgenograms are taken in the anteroposterior and the two oblique planes. Moderate downward pressure should be applied to the uterine fundus during exposure. Between the contrast margin of the fundus of the bladder and the fetal skull outline the total thickness of the translucent space should not exceed 6 to 8 mm. in normal cases. If this soft tissue gap is appreciably widened, it is almost certain that some abnormal structure is present in the uterine segment, the walls of which are very thin. This abnormal structure will be either a blood clot or a placenta praevia.—*S. G. Henderson.*

Braasch, William F., and Merricks, James W. Clinical and radiological data associated with congenital and acquired single kidney. *Surg., Gynec. & Obst.*, Sept., 1938, *67*, 281–286.

The article, after discussing certain clinical data associated with renal agenesia, emphasizes the more important roentgen data which are:

1. The outline of the solitary kidney is larger than the outline of the acquired single kidney or the normal kidney. The incidence of hypertrophy is twice as great and the degree of enlargement, when it is found, is greater.

2. The renal pelvis is frequently observed to be enlarged but the relative increase in size does not parallel the parenchymal hypertrophy. This is true in both congenital and acquired single kidney.

3. The renal shadow is usually found lower in congenital single kidney than in the acquired type.

4. The psoas muscle outline is less sharply defined on the side of the absent kidney in both congenital and acquired single kidney when compared with an unselected group of urograms.

5. There is an increase in the width of the psoas muscle on the side of the absent kidney in a few cases of agenesia (16 per cent of cases).

Because the roentgen shadow was normal or only slightly enlarged in 55 per cent of cases of acquired single kidney and in 20 per cent of cases of the congenital type, and because of the variability in the rate of hypertrophy of the remaining kidney after the removal of one, the surgeon cannot rely on the finding of a normal sized kidney at operation to prove the presence of a functioning kidney on the other side.

The cause for the poor definition of the psoas shadow on the affected side may be a matter of atrophy or some disturbance in innervation or lymphatic supply which existed between the psoas muscle and the absent kidney.—*P. C. Swenson.*

Jupe, M. H. Some observations on cases of suprarenal tumour. *Brit. J. Radiol.*, April, 1938, *11*, 242–251.

Primary tumors of the suprarenal gland which are rare compared with secondary deposits may be divided into two groups, cortical and medullary. Cortical tumors may be divided into adenomata and the so-called primary carcinoma of the suprarenal.

The primary carcinoma gives rise to an intra-abdominal mass, and a definite syndrome is seen. Usually, the tumor is found in female children under the age of ten, and gives rise to symptoms of virilism and obesity, producing a picture almost identical with Cushing's basophilism. Obesity with striae in the skin, hirsutes of male distribution, acne and other similar male manifestations may be seen. The urine may contain a male hormone, androsterone. The bones lack calcium as in basophilism.

Tumors of the medulla are generally referred to as neuroblastomata. In accordance with the degree of cell differentiation that has taken place the clinical picture tends to vary. The Pepper and the Hutchinson syndromes are described. The Pepper type affects very young children, the spread is confined to the abdomen, and the course of the disease is rapidly fatal. The first symptom in the Hutchinson type is commonly due to a metastasis, and the commonest site is under the pericranium. Other bones often involved are the ribs, sternum and long bones. An abdominal lump is frequently found. The bone metastases appear as areas of destruction, small and circumscribed, and usually widespread. Raising of the periosteum due to deposits of neoplasm under the periosteum is very common. The findings in 3 cases are given. The constant feature in these 3 cases is the raising of the periosteum of the femora. Congenital syphilis and hemopoietic conditions, such as leukemia and Cooley's anemia are accompanied by periosteal changes and must be considered in the differential diagnosis.—*S. G. Henderson.*

NERVOUS SYSTEM

Davison, Charles, and Wechsler, I. S. Erythroblastic (Cooley's) anemia and neurologic complications (status dysmyelinatus). *Am. J. Dis. Child.*, August, 1939, *58*, 362–370.

The authors report a case of erythroblastic anemia which conformed clinically to Cooley's case and subsequently reported cases but which differed from these in the neurologic manifestations which were present. These neurologic findings, such as the mask-like facies, lack of associated movements, pronator signs, hypertonicity and cogwheel rigidity were indicative of lesions in the extrapyramidal system. The status dysmyelinatus of the globus pallidus, the

pigmentary deposits about the ganglion and glia cells of the pallidum and striatum and the Alzheimer glia cells were undoubtedly responsible for the extrapyramidal symptoms. The loss in the usual pigment of the nerve cells of the substantia nigra and locus caeruleus most likely played no part, the authors believe, in the causation of neural signs. They regard the pathologic changes in the striatum and pallidum in their patient as somewhat analogous to those seen in hepatolenticular degeneration (Wilson's disease and pseudosclerosis).— *R. S. Bromer.*

SKELETAL SYSTEM

Anspach, William E., and Clifton, Willie Mae. Hyperparathyroidism in children. *Am. J. Dis. Child.*, Sept., 1939, *58*, 540–557.

Anspach and Clifton report 2 cases of hyperparathyroidism in children. The first case was that of a girl, nine years old at the onset of symptoms, who showed on roentgen examination generalized osteoporosis and metastatic calcification in the soft tissues of the right shoulder and knees. These changes were similar to those reported in hyperparathyroidism in older children and adults. Removal of a parathyroid adenoma relieved the condition and there was no recurrence of hyperparathyroidism during the subsequent three years.

The second case was that of an infant whose symptoms were first noted at the age of three months and progressed for two years. Blindness, thought to be due to increased intracranial pressure secondary to distortion and fractures of a soft skull, developed during the second year. Because of the hypercalcemia and hypophosphatemia, together with initial normal renal function, Anspach and Clifton believe that the diagnosis was primary hyperparathyroidism, despite the fact that the skeletal changes somewhat resembled those of renal rickets. The persistent loss of density in the endochondral zones in the roentgenograms indicates, they believe, that acalcification as well as decalcification may be a feature of primary hyperparathyroidism during active body growth.

During the period of observation prior to treatment, while there was persistent hypercalcemia and increasing decalcification, the serum phosporus value returned to normal. After roentgen irradiation of the neck, the bones recalcified. Polyuria and hypotonia were relieved. During the two years following irradiation of the neck there was no evidence of dysfunction of the parathyroids. The authors state that this is the youngest patient reported thus far and the only one they know who showed spectacular recalcification following roentgen therapy.—*R. S. Bromer.*

Oppenheimer, Albert. The swollen atrophic hand. *Surg., Gynec. & Obst.*, Oct., 1938, *67*, 446–454.

The author describes 14 cases of a peculiar swelling of the bones of the hand associated with atrophy of the skin and interosseous muscles. This syndrome could be correlated with unilateral abnormalities of the intervertebral foramina on the side of the affected hand. The nature of the lesion producing the bony constriction was not specific and did not vary the clinical syndrome. Only ultrashort wave therapy was used over the affected segments with a cure in 6 of 7 patients. The therapy produced no change in the roentgen findings so that the author explains the results as an action on the nerves producing a decongestion of their sheaths. Oblique roentgen projections of the cervical spine are essential to show the lesion.—*P. C. Swenson.*

Ecke, Walter. Zur Morphologie und Genese des Zwergwuchses; Untersuchungen an Liliputanern. (The morphology and genesis of dwarfism; examinations of Lilliputians.) *Fortschr. a. d. Geb. d. Röntgenstrahlen*, August, 1939, *60*, 107–134.

The studies were made on 11 Lilliputians, 9 males and 2 females from eighteen to forty-two years of age and all members of a dramatic troupe. They varied from 101 to 129 cm. in height. In the group were three pairs of siblings. Family histories revealed that the parents were all of normal size and there was no knowledge of dwarfism among the ancestors. The birth weights were all normal. In the majority of instances considerable growth in height occurred after the twentieth year. Numerous anthropologic measurements were made on 8 of the Lilliputians and tabulated. The proportions of the body were essentially normal though there was some tendency to brachycephaly and the weight tended to be high in proportion to the height when comparison was made with children of the same size. The facial features were usually childish and in none had

the voice changed from its childish character. The genitals were examined in only 2 males and they were observed to be disproportionately small in size and the testes were not palpable. The others gave evidence of extreme modesty and were not examined. There was a general absence of secondary sexual characteristics. The intelligence corresponded essentially to that of a normal adult and they were remarkably reliable in keeping appointments. Blood studies in 4 cases showed a tendency to lymphocytosis (41 to 58 per cent); electrocardiograms were normal in 5 cases.

Extensive skeletal studies with the roentgen ray showed a normal sella turcica in 8 cases and some enlargement over the normal standards of Kovacs in 5. The skeletal development shown in the hands, spine and pelvis corresponded to that of children between five and sixteen years of age. In 1 case there was shortening of the middle phalanx of the fifth finger which one commonly finds in mongolism, in another an enchondroma of a phalanx was discovered. In 8 of 11 cases there were definite changes in the femoral heads and pelvis resembling Perthes' disease. Aseptic epiphyseal necroses were also observed at the elbow and elsewhere. The development of the paranasal sinuses seemed more retarded than the rest of the skeletal development.

Koch has reported finding cystic degeneration of the hypophysis in a hereditary dwarf and changes have been observed in dwarfed animals. The cystic degeneration may be secondary to some congenital anomaly of the anterior lobe just as goiter forms in hypothyreosis. There follows a review of the literature and a discussion of the possible hypophyseal origin of the dwarfism in this group.—*W. A. Evans, Jr.*

MISCELLANEOUS

Meltzer, H., and Kühtz, E. H. Über den Ablauf einen experimentellen Entzündung unter der Wirkung der Röntgenbestrahlung. (The course of an experimental inflammation under the influence of roentgen irradiation.) *Strahlentherapie*, 1938, *62*, 425–435.

The authors attempted to determine the effect of roentgen irradiation on the salt picture occurring in the course of an inflammation. Meltzer has shown by previous experiments that in humans, rabbits and guinea pigs, an inflammation produces regularly a considerable displacement in the salts of the inflamed region, the nature of the displacement depending on the given stage in the course of the inflammation and is, therefore, characteristic of the age and of the condition of the inflammation. The method of study pursued by the authors was as follows:

Under aseptic conditions catgut sutures (oo) were introduced into the skin of the back of rabbits. Half an hour later the same skin region was irradiated with 825 r (or with fractional doses). After this, one suture with the neighboring skin was excised and examined.

Clinically the roentgen treated skin region differed in no way from a normal skin region into which a catgut suture is introduced under aseptic conditions. In all experiments the wound healed following the excision aseptically. The duration of healing was neither longer nor shorter than in the non-irradiated skin region and the operative scars differed in no way from those obtained in the non-irradiated region.

The hematoxylin-eosin stained histological sections presented a picture of a foreign body inflammation, the picture being dominated by edema and round cell infiltration. After the third day the catgut suture appeared encapsulated by a newly formed connective tissue. Of significance is the fact that on the fifth and sixth day, at a time which was later found to correspond with a definite decrease in the salt content all the phenomena of inflammation had regressed practically completely so that in the pathological sense the suture appeared to be situated in the tissue without practically producing any reaction and being surrounded only here and there by individual round cells and leukocytes, while on the seventh day the infiltration again increased.

Seven hours after the irradiation and after the introduction of the catgut suture, there occurs a definite increase in the salt content and this increase is uniformly distributed throughout the tissue. After this the ash content continues to increase, reaching its highest point on the third day. At this time there has formed a definite ring around the suture which shows an increased concentration of the salts directly around the suture. On the fourth day there occurs a decrease in the ash content and this decrease is most pronounced on the sixth day. At this time, however, the ash particles are again uniformly distributed throughout the skin re-

gion. On the seventh day the salt content begins to increase again.

The behavior of the calcium salt is parallel to the behavior of the total ash content. After seven hours the magnesium content is increased but is definitely decreased after fourteen and after thirty-two hours and again slightly increased on the second, fourth and seventh day. Only a part of the total magnesium salts consist of water insoluble compounds.

From the findings presented above, it is quite clear that under the influence of a single roentgen irradiation with 825 r the usual course of inflammation is completely changed. Similar changes occur also under the influence of fractional irradiation.—*A. S. Schwartzman.*

Langendorff, H. Das Verhalten des Retikulozytenwertes der weissen Maus nach Röntgenbestrahlung. (The behavior of the reticulocyte values of the white mouse following roentgen irradiation.) *Strahlentherapie*, 1938, *62*, 304–314.

According to Coutard the usual irradiation differs from the simple fractional method of irradiation basically by the decrease of the dose per time unit. While in the simple fractional irradiation the dose per minute corresponds to that of the intensive irradiation it equals in the protracted fractional method only about 2–2.5 r. Numerous communications have appeared in the literature regarding the mode of action of both forms of irradiation. In spite of that, even at present it is not quite clear whether the protracted fractional method of irradiation can be substituted by the simple fractional doses. In order to contribute to the solution of this problem, an attempt was made by this author to determine the effect of the various types of irradiation on the behavior of the reticulocyte values of the white mouse. The behavior of the reticulocyte value for purposes of determining the effect of the different types of roentgen irradiation was utilized because of the fact that previous studies have shown that the number of reticulocytes in the white mouse is usually constant or subject only to slight variations, and because of the fact that even a slight roentgen-ray dose is sufficient to produce a definite quantitative influence on the reticulocytes. A series of previous studies have shown that a single irradiation of a white mouse with doses of high intensity always produces a definite decrease of the young red cells found in the circulating blood. Depending on the size of the dose this phenomenon persists for one or more days after which the number of reticulocytes returns to normal. When the mouse is irradiated with doses of lesser intensity the time course of the reaction is changed in a given manner. After the administration of 100 r no demonstrable influence on the erythropoiesis is present; after the administration of 200 to 400 r, on the other hand, the course of the irradiation reaction takes a longer time when the protracted method is used in comparison with the simple method and a similar dosage. In these cases, therefore, the effect of the protracted dose is greater. A comparison of the curves obtained further shows that under a similar dosage, the radiation intensity increases the effect of the roentgen rays. This is applicable, however, only for intensities of 1 r per minute. If the dose per time unit, however, is below this value, then the time course of the reaction is the same in the two methods of radiation application.

When the dose of 1,000 r is divided in such a manner that individual fractional doses of a slight intensity are separated from each other by given intervals of time, then the effect of the roentgen rays on the reticulocyte values is dependent not only on the type of the time distribution and the height of the fractions, but also on the intensity of the radiation. If 100 r is given daily then at first no difference in the course of the two reaction curves is demonstrable after the irradiations. If the time interval is increased, however, between the individual irradiations, in that 100 r is given at intervals of two days, then the effect of the total dose on the hemopoietic tissue is considerably slighter than that obtained from irradiation with a higher intensity. Under these conditions the tissues get used to the irradiation and the effect is less pronounced.

On the basis of the results obtained, it may be stated that judging by the behavior of the reticulocytes, definite differences in the mode of action of the two methods of irradiation exist. The influence of the simple fractionated treatment on the tissue examined as well as on the entire body is always more pronounced than that resulting from the protracted fractionated irradiation. Nevertheless it must be emphasized that the course of the reaction after a long irradiation requires, in most cases, a greater time interval and that the same total dose is tolerated by the same type of animal to a better extent

than that following a simple fractionating of the dose.—*A. S. Schwartzman.*

Graham, J. Wallace. Radiation sickness; treatment with nicotinic acid. *J. Am. M. Ass.*, Aug. 19, 1939, *113*, 664–667.

In spite of increasing knowledge and improved technique in the use of roentgen rays, radiation sickness continues to be a major problem for those who are using high voltage therapy. The author reviews the literature on this subject. The cause and mechanism of the reaction are unknown and no evidence has been found to support theories regarding such etiologic factors as nitrogen retention and uremia, acidosis, alkalosis, liver damage, hypoglycemia, decreased blood cholesterol, retention of chlorides or injury to the intestinal epithelium. The diverse and sometimes contradictory theories with regard to etiology indicate that the mechanism of radiation sickness is still undefined.

Spies, Bean and Stone noted porphyrinuria in 5 of 7 cases of radiation sickness. Nicotinic acid in doses of from 200 to 1,000 mg. daily was administered to these patients, and the treatment was followed by a prompt cessation of nausea, vomiting, anorexia and headaches in all 7 cases and a return to normal of the urinary porphyrin. This drug was tried in a series of 70 cases under roentgen greatment at the Ontario Institute of Radiotherapy, Toronto General Hospital, including only those cases of radiation sickness in which severe nausea or nausea and vomiting were present. With the onset of the radiation sickness, patients were given 100 mg. of nicotinic acid twice or three times daily, either in the form of powder in half a glass of water and sipped slowly over half an hour, or in the form of 30 mg. tablets administered in a similar manner.

Following nicotinic acid therapy a result was classed as excellent if all symptoms were cured, good if vomiting stopped and some slight nausea remained, fair for cases in which vomiting was decreased to an occasional incident, and failure when the drug had little or no effect. The following table shows the results obtained:

	Result			
Area Irradiated	Excellent	Good	Fair	Failure
Abdomen	13	19	4	5
Chest	1	5	3	2
Head and neck	4	7	0	1
Inguinal gland	0	2	0	0
Head and abdomen	1	0	1	0
Chest and abdomen	0	0	2	0

The mode of action of nicotinic acid has not been determined, but no direct relation to porphyrin metabolism could be shown.—*S. G. Henderson.*

Baumeyer, S. Über die Wirkung Kurzwellenbehandlung auf maligne Tumoren. (The effect of short wave treatment on malignant tumors.) *Strahlentherapie*, 1938, *62*, 373–375.

Numerous attempts have been made to determine the effect of short wave therapy on malignant tumors. Even though there is no uniformity of opinion on the subject the results obtained by a predominant number of authors were negative.

Haas and Lob have found that short waves show no demonstrable effect on artificially inoculated malignant tumors. If the field strength is increased up to heat coagulation, the reaction of the tumors differed in no way from the reaction of the healthy tissue; these authors were therefore unable to demonstrate any specific biologic effect of the short waves on tumors. Practically identical results were obtained by Collier. These authors have received the impression that tumors treated with short waves showed a somewhat greater growth than nontreated tumors. Pflomm, on the other hand, reported to have obtained a disappearance of artificially inoculated sarcoma of rats following short wave treatment. At this point it must be mentioned that a spontaneous disappearance of sarcoma of rats is occasionally observed. In order to develop a hyperemia and thereby to intensify the sensitiveness of the tumors to roentgen rays, Fuchs subjected to the short wave treatment 2 patients suffering from pulmonary carcinoma prior to the treatment. He believes to have improved somewhat the results obtained in this manner. Urbach and others, on the other hand, advise against the use of short wave treatment in malignant tumors.

This author had an opportunity to study the effect of short wave treatment on a patient and the results obtained by him are summarized briefly as follows:

1. Short waves have a definite influence on malignant tumors.

2. Simultaneous short wave and roentgen irradiation may lead to an enlargement of malignant tumors.

3. A "sensitization" of tumors to roentgen rays is not obtainable by short waves.

4. Short wave treatment of malignant tumors must be warned against.—*A. S. Schwartzman.*

Smith, Lawrence W., and Fay, Temple. Temperature factors in cancer and embryonal cell growth. *J. Am. M. Ass.*, Aug. 19, 1939, *113*, 653–660.

This paper presents clinical, pathological and biological evidence that "temperature" plays one of the most important parts in the activation of embryonic cell growth. Two common principles apparently apply to cellular activity, embryonal cell development and plant germination.

1. Favorable temperature is the chief activator in cell growth.
2. The physical spectrum (from roentgen rays to ultraviolet rays) contains some quality which is inhibitory to cellular growth and regulates maturation and differentiation.

Initial experiments were designed with the simple incubation of fertile eggs at temperatures ranging from 108 to 90°F. for the twenty-one day period usually necessary for the normal hatching of the chick. There is a narrow range between 97 and 104°F., which might be spoken of as the optimal zone, in which normal growth, differentiation and organization of the tissues take place, with the development of a normal chick. At the lower level there is a critical temperature lying at approximately 94 or 95°F., at which there will be marked retardation in the development of the embryo and a viable chick will only rarely develop. It is the author's impression, gained from chick experiments, that a temperature just above freezing is not as seriously detrimental to the later normal development of the embryo as is the maintenance of a temperature just below the critical level. In the attempt to demonstrate the importance of temperature with respect to embryonic cell growth, a series of experiments was undertaken, in which the effect of temperature on the growth of normal and cancer cells was studied by tissue culture methods. Preliminary observations bear out the theory that critical levels exist for these cells and that tumor cells are more readily affected by alteration in temperature than are normal, differentiated, adult type cells. The importance of heat and light with respect to germination, growth, differentiation and organization of plants is mentioned.

The investigation of the effect of alteration in temperature on the growth of immature, undifferentiated neoplastic cancer cells in human beings was the next step in this work. Thirty-eight patients with inoperable cancer have been treated by local refrigeration alone combined with artificial "hibernation," in which for periods as long as five days the body temperature as read by rectal thermocouple was maintained at levels as low as 85 or 90°F. By refrigeration is meant the application of cold locally by means of ice water or a brine solution circulated through an apparatus designed especially to fit the structures in which the lesion exists. By hibernation is meant a general reduction of body temperature below the critical level of 95°F. Through the withholding of food, complete exposure of the body to a constant low environmental temperature (50 to 60°F.) maintained by an air conditioning unit, and light narcosis produced with small doses of avertin with amylene hydrate and of barbiturates, a state of artificial "hibernation" can be maintained in which metabolism is reduced to an almost negligible figure, with arrest of bowel and renal function. During such hibernation neoplastic lesions are diminished in size even more strikingly than by refrigeration alone. In every instance there has been a prompt reduction in pain. There has been regularly a rapid, gross, measurable decrease in the size of the lesion within twenty-four to forty-eight hours. There has been also a general improvement in the patient's condition, with a gain in weight, a better appetite and a change for the better in the mental state. These patients had all been given up from the standpoint of the more orthodox methods: operation and irradiation.

Serial biopsy specimens were taken at varying intervals after the initiation of local refrigeration therapy. Within the first twenty-four to forty-eight hours there occurs a persistent ischemia, the effect of continuous cold. Within the first forty-eight hours definite changes in the staining capacity of the tumor cells are frequently found. Within the next few days a degenerative change takes place which ranges from the minimal changes already noted to frank necrosis and complete disintegration of the cells. So long as the temperature locally

is not reduced below an arbitrary figure of 40°F. no demonstrable changes take place in the overlying normal adult differentiated structures such as the skin and subcutaneous tissue.

The authors believe that the application of subcritical temperatures, through methods of local and general "refrigeration," may offer a valuable therapeutic adjunct to our present method of treatment of undifferentiated cell growth, particularly of carcinoma.—*S. G. Henderson.*

Carty, John R., and Ray, Bronson S. Some experiences, experimental and clinical, with direct irradiation of neurological tumors during operation with low voltage radiation. *Radiology*, March, 1939, *32*, 325–331.

Avoiding any controversy of the relative value of high voltage and low voltage therapy in cases of brain tumor, the author reports his experience with very low voltage high intensity irradiation in experimental animals and in 12 patients suffering from brain or cord tumors.

The apparatus used was a shock proof machine equipped with various sized cones which could be sterilized. The inherent filtration of the oil in the tube was equivalent to 0.5 mm. Al and the intensity was such that 2,000 r measured in air could be given in twenty minutes at a distance of 20 cm.

Experiments were conducted by irradiating the brains of normal dogs through a window 2 cm. in diameter cut in the parietal bone. Various amounts were given and although all the experiments are not completed it appears that the dog's brain can stand dosages up to 12,000 r at low voltage without showing clinical effects or more than minor microscopic changes. Using larger dosages of 18,000 r it was possible to demonstrate that the depth intensity of the rays is limited and there is comparatively little scattering effect outside the limits of the direction of the beam.

Six cases of brain tumor and 6 of cord tumor, all inoperable, were treated by localizing the beam to the residual tumor as soon after a radical excision as possible; 2,000 r at 60 kv (peak) at 20 cm. distance was given. The only immediate reactions noted were an unexplained fever in 2 of the cord tumor cases and 1 of the brain cases and a temporary facial palsy developed in 1 brain case.

Sections taken from the tissue in the center of the roentgen-ray beam showed definite tumor cell destruction within twenty minutes after the beginning of the irradiation.

This method of treatment is so new that no conclusions can be drawn as to its ultimate value. In discussing this paper, Dr. Sherwood Moore said, judging from his own experience, he was sure that a great deal of good could be accomplished by this form of irradiation in hopeless cases.—*J. H. Harris.*

Bromley, J. F. Low-voltage, near-distance x-ray therapy. *Brit. J. Radiol.*, May, 1938, *11*, 289–296.

Author's Summary:

1. Low-voltage short-distance irradiation has a valuable place in radiotherapy. The limitations of the method must be appreciated, and these are implied in the title. In order to emphasize the essential principle of very short distance, the term "contact" therapy, which was at one time used, would seem to be permissible, even if not strictly accurate. The strict localization of the beam, both laterally and deeply, is an advantage in numerous situations.

2. Cooperation with the surgeon will increase the sphere of usefulness for the tube, particularly in mouth and rectal cancer, and possibly in some cases of cancer of the lung.

3. Full dosage, founded on careful preliminary measurements, is essential. Administration of a series on an area which has received a previous dose which has proved inadequate is both dangerous and liable to failure. At least 6,000 r must be given to carcinomatous tumors. For rodent ulcer, smaller doses are permissible.

4. Multiple small fields directed to the periphery of a tumor are, in general, more efficient than single larger fields. Occasionally, small single fields are permissible in treating rodent ulcer in the canthus of the eye and similar situations.

5. An interesting line of research is made possible by the ease with which the beam can be localized to an isolated node of skin cancer, or to a particular portion of tumor.—*S. G. Henderson.*